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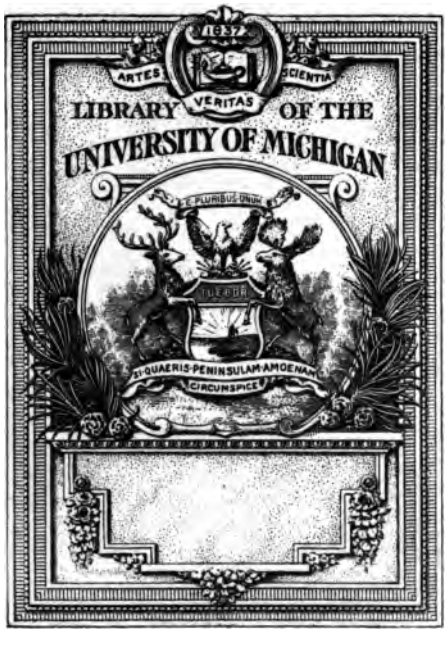
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OF

LONDON.

NEW SERIES. VOL. X.

SESSION 1890-91.

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c. 1888. Hirsch, A., M.D., 113, Pottsdamer Strasse, Villa 2, Berlin.
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1888. Koch, R., M.D., 36, Klosterstrasse, Berlin.
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c. 1888. MacLaurin, H. A., M.D., Macquarie Street, Sydney, N.S.W.
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1852. Richardson, B. W., M.D., F.R.S., 25, Manchester Square, W.
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N.R. 1883. Kirker, Gilbert, R.N., M.D., R. N. Hosp., Gosport.
1881. Kynsey, W. R., Colombo, Ceylon.
C. 1857. Leas, Jose Fernandes da Silva, Cape de Verde.
C. 1875. Lent, —, M.D., Cologne.
C. 1886. Le Roi de Méricourt, L., M.D., 5, Rue Cambacères, Paris.
N.R. 1879. Lilburne, James, M.D., Dep. Inspec.-Gen., Duncrivie House, Milnathort, N.B.
1886. Lingard, A., Poonah, India.
C. 1851. Litt, W., Veterinary Surgeon, Shrewsbury, Salop.
N.R. 1873. Little, James, M.D., 14, Stephen's Green North, Dublin.
N.R. 1885. Lloyd, W. Harris, M.D., Dep. Inspec.-Gen. (*gone abroad*).
C. 1857. Lowtzoff, —, St. Petersburg, Russia.
N.R. 1881. Lyons, R. T., M.D., Surgeon-Major, Rampart Barracks, Fort William, Calcutta.
C. 1887. McLaurin, H. A., M.D., Sydney, New South Wales.
C. 1870. Mallet, —, M.D., Warsaw.
N.R. 1867. Marshall, John, 13, Liverpool Street, Dover.
N.R. 1860. May, George, jun., 43, Castle Street, Reading, Berks.
N.R. 1889. McVail, John C., M.D., Kilmarnock, Ayrshire.
N.R. 1890. M'Neill, R., M.D., Argyll County Council, Oban, N.B.
C. 1886. Moleschott, Jacob, M.D., Rome.
N.R. 1872. Moore, John Wm., M.D., 40, Fitzwilliam Square West, Dublin.
C. 1851. Monzon, Rafael, Barbacaos, New Grenada.
C. 1853. Neumann, —, M.D., Berlin.
C. 1851. Noble, Daniel, M.D., 258, Oxford Road, Manchester.
N.R. 1881. Oldham, Chas., Brigade Surg. Ind. Med. Dept., Shurdington Road, Cheltenham.
C. 1872. Poggio, Don Ramon Hernandez, Cuba.
C. 1854. Purple, S. S., M.D., New York, U.S.A.
N.R. 1879. Ransom, W. H., M.D., F.R.S., Nottingham.
N.R. 1875. Ransome, Arthur, M.D., F.R.S., Devisdale, Bowdon, Cheshire.
N.R. 1884. Ray, George, Surg.-Gen., 76, Jermyn Street, S.W.
N.R. 1880. Reid, Sir J. Watt, K.C.B., M.D., R.N.
N.R. 1874. Reid, Walter, M.D., Fleet-Surgeon, Royal Naval Hospital, Haslar.
N.R. 1866. Robertson, James, M.D., Alfred Place, Melbourne.
C. 1886. Rochard, G., M.D., Paris.
N.R. 1863. Rowe, T. Smith, M.D., Cecil Street, Margate.
N.R. 1879. Ross, J. T. C., C.I.E., Surgeon-General Ind. Med. Dept., The Grove, Ryde, Isle of Wight.

Elected.
N.R. 1881. Russell, E. G., M.B., B.Sc., Surg. Ind. Med. Dept., Gauhàti, Assam, India.
N.R. 1875. Russell, J. B., M.D., 1, Montrose Street, Glasgow.
N.R. 1859. Sanderson, J. Burdon, M.D., F.R.S., 64, Banbury Road, Oxford.
C. 1851. Sankey, F. F., Malta.
C. 1879. Sarell, Richard, M.D., Pera and Therapia, Constantinople.
C. 1873. Schleisner, —, M.D., Copenhagen.
C. 1874. Schlimmel, —, M.D., Teheran.
C. 1852. Schürmayer, Professor, Heidelberg, Germany.
N.R. 1858. Scratchly, G., New Orleans, U.S.A.
C. 1851. Sharpe, Richard, Beverley Road, Hull.
C. 1853. Sigmund, C. L., Vienna.
N.R. 1884. Simpson, W. J., M.D., Bengal Club, Calcutta.
N.R. 1888. Smith, F., Montreal, Cargate Avenue, Aldershot.
1890. Smith, P. C., Motherwell, Lanarkshire.
N.R. 1867. Smith, R. Wagstaff, Mount Rundell, Harborne, Birmingham.
C. 1886. Sonsino, Prospero, 11, Via San Lorenzo, Pisa.
N.R. 1854. Spinks, C. N., Sankey Street, Warrington.
C. 1851. Spooner, W. C., Vet. Surg., Southampton.
C. 1876. Sternberg, Geo. M., M.D., United States Army, Port Point, San Jose, San Francisco, California.
N.R. 1889. Street, A. F., M.D., Westgate-on-Sea.
N.R. 1885. Sturridge, P. F., Kendal, Westmorland.
N.R. 1880. Tatham, John, M.D., Town Hall, Salford, Manchester.
C. 1860. Taylor, H. Sharp, 15, Quarry Street, Guildford.
C. 1855. Tholozan, J. Désiré, M.D., Teheran.
C. 1888. Thompson, Ashburton, M.D., Sydney, New South Wales.
N.R. 1853. Todd, J. George, Evenwood, West Auckland, Durham.
N.R. 1885. Tomes, Arthur, Surg. Ind. Med. Service, Bengal.
N.R. 1873. Townsend, Stephen Chapman, C.B., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner for Central India.
C. 1851. Tribes, E., Nismes, France.
C. 1855. Tribuchet, Adolphe, Paris.
C. 1853. Valerj, —, M.D., Rome.
C. 1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
C. 1856. Von Iffland, —, Beaufort, New Quebec.
N.R. 1879. Watson, Alexander, M.D., R.N., Dep. Insp.-Gen., Army and Navy Club.
C. 1853. Watson, John, M.D., Jamaica.
N.R. 1887. Welch, Henry, M.D., Blackpool, Lancashire.
N.R. 1880. Whishaw, J. C., M.D., Surg.-Major Ind. Med. Dept., Lucknow.
N.R. 1880. White, J. Berry, M.D., Surg.-Major Ind. Med. Dept. (*address not known*).
C. 1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.
N.R. 1854. Wiblin, John, Southampton.
N.R. 1865. Williams, C., 48, Prince of Wales's Road, Norwich.
N.R. 1884. Williamson, R. E., M.B., Brookfield, Lymm, Cheshire.
N.R 1875. Wortabet, John, M.D., St. John's Hospital, Beyrout.

TRANSACTIONS

OF THE

Epidemiological Society of London.

A BRIEF REVIEW OF RECENT SANITARY LEGISLATION.

INAUGURAL ADDRESS OF SESSION 1890-91.

BY ALD. JOSEPH EWART, M.D., F.R.C.P., J.P., PRESIDENT.

(*Read : November* 19*th*, 1890.)

MY first duty is to thank the Society most cordially for having elected me to preside over its affairs during the present official year. The office of President is one of equal honour and responsibility. To maintain its dignity unsullied and to conserve its interests undiminished shall be my constant endeavour. It will, however, be apparent that its prosperity is mainly in the hands of its members—especially those who, having the necessary leisure, may be good enough to contribute papers for consideration and discussion. If, in this respect, the experience of the past proves to be a tolerably fair measure of the history of the session now in process of inauguration, there will be furnished ample reason for congratulation. That this expectation at least may be fully realised—nay exceeded, if it is possible to do so—is devoutly to be wished.

When we survey the enormous strides which have been made during the past two generations in all departments of literature, science, art, industry, and medicine—curative and preventive—particularly in epidemiology, and its handmaidens, hygiene, sanitation, and sanitary legislation, it is reasonable to anticipate that the pace will be quickened in the coming time rather than slowed or decreased. When it is further borne in mind that the welfare of our race and that of the lower animals essentially useful to mankind are largely dependent upon the uninterrupted extension of our knowledge regarding the etiology and natural history

of infectious and communicable diseases, and the imperfectly understood conditions which, from time to time, facilitate their diffusion in an epidemic form, we may rest assured that the mere consciousness or recognition of these things will be sufficient to stimulate contributors to achievements in the domain of epidemiology—equally honourable to themselves, creditable to their profession, and beneficial in the highest degree to humanity in its widest acceptation.

It is true that we know much more about spreading diseases now than we did in the decade prior to the beginning of the Victorian era. True it is also that, during that period, we have had to unlearn a good deal. The net result of this learning and unlearning has been to clear the ground, to mark progress in the right direction, and to secure greater exactness of the knowledge acquired. This is specially apparent in the advance of both human and comparative physiology; in the rapid development and growth of bacteriology, meteorology, electricity, and magnetism; the abrogation of the old-fashioned system of quarantine, and the wide-spread adoption, in practice, of the principles of hygiene and sanitation in dealing with the accepted causes of infectious, communicable, and epidemic disease.

But the more we succeed in unravelling, by way of observation, experiment, or reseach, the secrets of Nature, in their relations to avoidable or preventable disease, the more we shall find remaining to be investigated. In this conception we are reminded of the modest declaration of the immortal Newton, viz., "I do not know what I may appear to the world, but to myself I seem to have been only like a boy playing on the sea-shore, and diverting myself in now and then finding a smoother pebble, or a prettier shell, than ordinary, whilst the great ocean of truth lay all undiscovered before me." In modern times, many smoother pebbles and prettier shells than ordinary, throwing a bright light on the etiology of infectious diseases, both in their endemic and epidemic forms, have been found. Still, like Newton, we are only on the threshold of the subject, whilst the great ocean of truth lies unexplored before us.

It seems to me that, so far as our knowledge of epidemiology extends, it has been ably and exhaustively handled in this Society during the last few years, and recorded in its *Transactions*. I propose, therefore, that the short time allotted to me for the delivery of my address, be devoted to a brief review of some recent legislation, largely designed to curtail the spread of preventable diseases, or, as far as may be, to stamp them out altogether. The study has been dull

and dry, but not unprofitable. None of the measures, springing out of this legislation, can be regarded as being fairly abreast of the advanced science and social economics of the day.

These Acts of Parliament have, speaking generally, been framed with the design of ameliorating the condition of considerable sections of the community, and through these, the entire population. Though not nearly so advanced and comprehensive as many would justly wish to see them, they are nevertheless based upon lines easily susceptible of such expansion as will meet the demands and necessities of the future. The good intentions displayed in their construction, for which the proverbial praise may be accorded, are, however, materially frustrated by the cumbrousness and costliness of their provisions, not to mention legal subtleties, formulæ, and forms retained in full force, even for the benefit of owners of houses acknowledged to be insanitary. To such an extent is this carried still by the law as it stands, that the so-called rights of property are held to be sacred, and, accordingly, take the first place, whilst the claims of the humbler, helpless, and poorer classes of humanity are relegated to a secondary position. That property has duties as well as rights is not yet adequately recognised.

As an important means of diminishing the diffusion of, and mortality from, infectious disease, may be cited the widespread adoption of the *Infectious Disease (Notification) Act* of 1889. After the expiration of two months from the passing of the Act, it was extended to every district in London. Its adoption by "any urban, rural, or port-sanitary district" is permissive. The dual system of notification is to be enforced. Thus Section 3 (1) directs that, "Where an inmate of any building used for human habitation within a district to which this Act extends is suffering from an infectious disease to which this Act applies, then, unless such building is a hospital in which persons suffering from infectious disease are received, the following provisions shall have effect, that is to say:—(*a*) The head of the family to which such inmate (in this Act referred to as the patient) belongs, and in his default the nearest relatives of the patient present in the building, or being in attendance on the patient, and, in default of such relatives, every person in charge of, or in attendance on, the patient, and in default of any such person, the occupier of the building, shall, as soon as *he becomes aware* that the patient is suffering from an infectious disease to which this applies, send notice thereof to the Medical Officer of Health of the district; (*b*) every medical practitioner attending on, *or called in* to visit, the

patient, shall forthwith, *on becoming aware* that the patient is suffering from an infectious disease to which this Act applies, send to the Medical Officer of Health for the district a certificate stating the name of the patient, the situation of the building, and the infectious disease from which, in the opinion of such medical practitioner, the patient is suffering."

It will be noted that the medical attendant is not called upon to notify until he has satisfied himself that the patient is the subject of an infectious disease, and that to ensure the greatest accuracy in diagnosis practicable a second opinion may be obtained. Section 2 of Clause 4 enacts that the local authority "shall pay to every medical practitioner for each certificate duly sent by him in accordance with this Act, a fee of two shillings and sixpence if the case occurs in his private practice, and of one shilling if the case occurs in his practice as medical officer of any public body or institution." That covers payment of the fee to any and every practitioner who may have certified in any given case, according to law.

By Sec. 2 it is directed that "a payment made to any medical practitioner in pursuance of this Act shall not disqualify that practitioner for serving as member of the Council of any county or borough, or as a member of a sanitary authority, or as guardian of a union, or in any municipal or parochial office." Where the Officer of Health is in attendance upon a patient, "he shall be entitled to the fee to which he would be entitled if he were not such medical officer." This provision is as important as it is liberal.

Section 6 defines the infectious diseases to be notified, namely, small-pox, cholera, diphtheria, membranous croup, erysipelas, scarlatina or scarlet fever, typhus, typhoid enteric, relapsing, and puerperal fever, and *any other* infectious disease to which the Act has been applied by the local authority. Power is given, in Section 7 and its sub-sections, to the local authority to extend the definition of infectious disease.

In an exhaustive paper, entitled *The Bearing of School Attendance upon the Spread of Infectious Diseases*, by Dr. A. Newsholme, the distinguished Medical Officer of Health at Brighton, read at the late Congress of the Sanitary Institute, held there in August last, the question of including whooping-cough and measles among the notifiable diseases was discussed. The author adduced statistical and other evidence to show that these diseases should be notified, like scarlet fever and diphtheria, and doubted whether there would be any necessity for the closure of schools, owing to the presence of

infectious disease among the scholars, excepting when insanitary conditions in the schools themselves had given rise to disease. As regards whooping-cough, its existence in a house should at least preclude all the children of the same house from attending infant schools. As matters now stand with regard to measles—in the absence of notification, it frequently happens that an epidemic of this highly-infectious disease seriously reduces the average attendance of a school before the Medical Officer of Health is fully apprised of its existence and prevalence. He declares that its rapid spread is "largely due to ignorance of its presence and to the fact that we have hitherto scarcely attempted to cope with it. We have, in scarlet fever, an instance of what preventive measures can effect; if measles be more difficult to deal with, owing to its infectiousness before the rash appears, let us not be daunted by the task, but do our utmost to secure complete and prompt information in regard to it, and we shall then have done much to add it, as well as whooping-cough, to the list of diseases which are becoming steadily diminished by sanitary measures."

The readiness with which the provisions of this Act have been adopted throughout the country is most encouraging. It shows that the education which has been going on with regard to hygiene and sanitation among the burgesses and their representatives during the past fifty years or so has been as widepread as it has been effective. The result will probably be a continued extension of this progress at an accelerated pace. Before the passing of this Act, fifty-six Local Authorities had obtained special powers authorising compulsory notification of infectious disease in their respective districts, covering a population of 3,750,000. The Act has been adopted since it was passed, little more than fourteen months ago, by local bodies representing twelve millions of souls, whilst it was compulsorily enforced among the four millions comprising the population of London; so that it has already been applied to about 20,000,000, or nearly four-fifths of the population of England and Wales.

Beyond making the Health Officer better acquainted with the geographical distribution of infectious and epidemic disease in his district, the Act has not materially altered his legal position. Armed, however, with this increased information, he will now be prepared, as he could not be before, with unanswerable arguments to enable him to impress upon his employers the imperious necessity of making ample and prompt arrangements for the reception and treatment of all cases of infectious disease, which cannot be properly cared for in their

own homes, and others demanding such management in a public institution specially adapted for the purpose. Already those towns which have been imperfectly provided with the necessary hospital accommodation are taking steps to secure it without further delay.

When notification, with such means of isolation, has become universal, either by the further adoption of the permissive clauses of the Act, or, this failing, through its being made compulsory everywhere, we may confidently anticipate, in the near future, a marked decrease in the prevalence of, and mortality from, infectious disease. The earlier intimation of isolated outbreaks will also tend to the more prompt introduction of the provisions of the Public Health Act, and the consequent correction of discovered sanitary defects—an outcome of notification—must greatly contribute to the improvement of the public health. This, too, notwithstanding the fact that the new Statute in no way increases the direct powers or authority of the Officer of Health. It is manifest that the full fruition of the Act will not be realised until it has been applied throughout the length and breadth of the land. This accomplished, the sympathetic co-operation of the medical practitioners with the Medical Officers of Health will soon do the rest, and bring the working of the system as near to perfection as possible.

The next measure to which I propose to invite your attention is the *Infectious Disease (Prevention) Act*, passed on the 4th of August 1890, or an Act to Prevent the Spread of Infectious Disease. Its provisions extend to every London district after the expiration of four months from the passing of the Act, and to any urban or rural sanitary district after the adoption thereof; and the Local Authority of any urban or rural sanitary district may adopt all or any of the sections of this Act, by a resolution passed at a meeting of such authority. The procedure required to confirm this resolution is described (Sect. 3). The power of the local body to rescind any resolution adopting all or any of the sections of the Act, either wholly or as regards any of the adopted sections, is briefly detailed in Section 21. It will be perceived that, outside London, its entire or partial adoption is permissive.

Roughly, the Act may be split into two divisions, namely: (*a*) provisions designed to protect the consumer from tainted or infected milk; (*b*) and the public against several sources of infection. It has long been felt that the law was defective as regards the supply of milk in towns. It is true that, in some instances, urban Sanitary Authorities have been able to deal effectually, in suspected cases, with milk sellers

within their jurisdiction by powers provided in local Acts. For example, the Medical Officer of Health for Brighton is empowered by the Town Improvement Act to enter suspected dairies, institute inquiries, and to command the production of a list of customers. In one instance, occurring within my own cognizance, the prompt exercise of the provisions of this statute was signally successful in completely nipping in the bud what must otherwise have proved to be a disastrous milk epidemic of typhoid fever.

It has also been felt that, if the Medical Officer could not be authorised to examine cattle reported to be suffering from disease, he ought, at any rate, to have authority to secure the services of a qualified veterinary surgeon to furnish him with reliable information on the subject. This defect is repaired by this statute. Thus, in case the Medical Officer of Health is in possession of evidence that any person in the district is suffering from infectious disease, attributable to milk supplied within the district from any dairy situate *within* or *without* the district, or that the consumption of milk from such dairy is likely to cause infectious disease to any person residing within the district, such Medical Officer shall, if authorised in that behalf by an order of a justice having jurisdiction in the place where such dairy is situate, have the power to inspect such dairy, and if accompanied by a veterinary inspector, or some other properly qualified veterinary surgeon, to inspect the animals therein, and if, on such inspection, the Medical Officer of Health shall be of opinion that infectious disease is caused from consumption of the milk supplied therefrom, he shall report thereon to the Local Authority, and his report shall be accompanied by any report furnished to him by the said veterinary inspector or veterinary surgeon, and the Local Authority may thereupon give notice to the dairyman to appear before them within such time, not less than twenty-four hours, as may be specified in the notice, to show cause why an order should not be made requiring him not to supply any milk therefrom within the district until such order has been withdrawn by the Local Authority, and if, in the opinion of the Local Authority, he fails to show such cause, then the Local Authority may make such order as aforesaid; and the Local Authority shall forthwith give notice of the facts to the Sanitary Authority and County Council (if any) of the district or county in which such dairy is situate, and also to the Local Government Board.

When the Medical Officer or the Local Authority is satisfied that the milk-supply has been changed, or that the cause of infection has been removed, the prohibitory order shall be

forthwith withdrawn. Refusal to permit the Medical Officer, "on the production of an order from a justice to inspect any dairy, or if so accompanied, as aforesaid, to inspect the animals kept there, or supplying milk within the district, in contravention of the order forbidding the supply or sale of the same within the district, is to be deemed as an offence against the Act." Proceedings, however, in respect of such offence shall always be taken before the justices of the peace having jurisdiction in the place where the said dairy is situate. Provision is also made that no dairyman shall be liable to an action for breach of contract if the breach be due to an order from the local authority under this Act (Sect. 4).

Provisions are thus made (1) to empower an Officer of Health to inspect a dairy, and at the same time to have the animals examined and reported upon by a veterinary surgeon; (2) to interdict the supply of milk suspected of being infected or produced by unhealthy cows; (3) to secure the prosecution of contumacious dairymen; (4) and to enable him to put these powers in motion, in a rather summary manner, either *within* or *without* his jurisdiction. The careful and energetic administration of this portion of the statute, as occasion may demand, will doubtless tend to prevent the spread of typhoid fever, scarlatina, diphtheria, and tuberculosis, and, it may possibly be, other diseases disseminated by the consumption of infected milk.

The other sections of the Act, intended to protect the public against various common sources of infection, are very important. They comprise summary powers: (*a*) for the cleansing and disinfection of infected premises, rooms, bedding, and clothing; (*b*) prohibiting the undue retention of the dead bodies of persons who have died of any infectious disease; (*c*) the removal of bodies of persons, who have died of infectious disease in hospitals, only for burial; (*d*) to authorise justices in certain cases to order dead bodies to be buried; (*e*) the purifying of public conveyances used for carrying infected corpses; (*f*) the detention of an infected person without proper lodging in hospital by the order of a justice; (*g*) avoiding the throwing of infectious rubbish into ashpits without being previously disinfected, all of which have a direct bearing on the improvement and conservation of the public health.

The Act to which I now propose to call attention is *The Housing of the Working Classes Act*, 1890, which came into operation on the 18th August last. It does not amend the existing laws on the subject to any considerable extent. Its chief object is to place before the public and Local Authorities,

in an accessible form, thirteen Acts, and portions of two other Acts, bearing upon the housing of the working classes, passed between 1850 and 1885, including Torrens's and Cross's Acts. Some amendments have been made, but the leading feature is handiness and consolidation of the law. The subject is divided into several parts: (*a*) unhealthy areas; (*b*) unhealthy dwelling houses; (*c*) working-class lodging houses; (*d*) supplemental; (*e*) application of Act to Scotland; (*f*) application of Act to Ireland; (*g*) repeal and temporary provisions. The purpose I have in view will be sufficiently met by a concise notice of the first three divisions of the Act:—

Part I.—Unhealthy Areas.

This portion is practically a re-setting of the provisions of Lord Cross's Artisans' Dwellings Act of 1875, and its Amending Acts. It authorises (1) that where an official representation to the effect that "within a certain area" (*a*) any houses, courts, or alleys are unfit for human habitation, or (*b*) the narrowness, closeness, and bad arrangement, or the bad condition of the streets and houses, or groups of houses, within such area, or the want of light, air, ventilation, or proper conveniences, or any other sanitary defects, or one or more of such causes, are dangerous or injurious to the health of the inhabitants, either of the buildings in the said area, or of the neighbouring buildings, and that these evils cannot be remedied otherwise than by the re-arrangement and reconstruction of the streets and houses within such area—the substitution of wholesome dwellings for the working classes in place of those removed by an improvement scheme.

The Local Authority must take such representation into consideration, and if satisfied of its truth and of the sufficiency of their resources, "shall pass a resolution to the effect that such area is an unhealthy area, and that an improvement scheme ought to be made in respect of such area, and after passing such resolution, they shall forthwith proceed to make a scheme for the improvement of such area." (Sect. 4.)

The procedure as to the commencement of proceedings, the preparation and confirmation of the scheme, and the provision of dwelling accommodation for the persons displaced, are the same as in the old Acts. (2) It provides for an improvement scheme to be drawn up by the Local Authority and confirmed by the Local Government Board and Parliament; (3) for the execution of the scheme, as soon as practicable, by the purchase of land within the area by agreement or compulsorily; (4) for the clearance, re-arrange-

ment and re-laying out of the land; (5) for the re-letting or re-sale of part, parts, or the whole of the land, on conditions fixed by the Local Authority; (6) for the re-building of dwellings and other works by contractors, or, where special permission has been granted by Parliament, even by the Local Authority, subject in this latter case to sale within a period of ten years.

The official representation is made by the Medical Officer of Health whenever he sees cause to make the same; but, on receiving a complaint from two or more justices of the peace acting within the district for which he acts as Medical Officer of Health, or from twelve or more ratepayers, of the unhealthiness of any area within such district, it shall be the duty of the Medical Officer of Health forthwith to inspect such area, and to make an official representation stating the facts of the case, and whether in his opinion, the said area, or any part thereof, is an unhealthy area, or is not an unhealthy area. (Sect. 5.) Should he fail to do so, the ratepayers may appeal to the Local Government Board.

According to Cross's Act, as many persons of the working-class as may be displaced in the County or City of London must be accommodated either *within* the area to be dealt with, or in the *immediate vicinity*. But in this Act it is enacted that when the confirming authority has been satisfied that equally convenient accommodation "can be provided for any persons of the working-classes displaced by the scheme at some place other than within the area, or the immediate vicinity of the area comprised in the scheme," and that it "has been, or is about to be, forthwith provided, either by the Local Authority or by any other person or body of persons," the confirming authority may authorise such an arrangement "to the extent to which accommodation is so provided." (Sect. 11, *a*.)

Under certain special circumstances of locality, and "to the number of artisans and others belonging to the working-class dwelling within the area and being employed within a mile thereof," the confirming authority may deem it expedient to "dispense altogether with the obligation of the Local Authority to provide for the accommodation of the persons of the working-class who may be displaced by the scheme to such extent as the confirming authority may think expedient, but not exceeding one-half of the persons so displaced." (Section 11, *b*.) Elsewhere than in London, the confirming authority may "provide for the accommodation of such number of those persons of the working-classes displaced in the area with respect to which the scheme is

proposed *in suitable dwellings*, to be erected in such place or places, either within or without the limits of the same area, as the said authority, on a report made by the officer conducting the local inquiry, may require." (11 Sect. 2.)

Elaborate and lengthy provisions are made for the acquisition of the land. The estimate of value of such lands and interest shall be based upon *the fair market value* at the time when the valuation was made, strict regard being had to the nature and the condition of the buildings, their probable duration in their existing state, and their state of repair. An important proviso is that *no additional allowance is to be made by the arbitrator in respect of compulsory purchase.* Further, no addition or improvement of the property made after the date of publication of the scheme shall be included in the estimate, unless necessary for the maintenance of the same in a proper state of repair; neither shall any interest acquired after said date be allowed to increase the amount of compensation to be paid. (Sect. 21.)

Where the arbitrator has been satisfied that (*a*) the rental of a house or premises was increased by reason of the same being used for illegal purposes, or so over-crowded as to be dangerous to the health of the inmates, the compensation shall, so far as it is based on rental, be based on the rental which would have been obtainable if the house or premises were occupied for legal purposes, and only by the number of persons whom the house or premises were, under all the circumstances of the case, fitted to accommodate without such overcrowding as is dangerous or injurious to the health of the inmates; (*b*) that the house or premises are a nuisance, or are in a state of defective sanitation, or are not in reasonably good repair, the compensation shall be the amount estimated as the value of the house and premises if the nuisance had been abated, or if they had been put into a sanitary condition, or into reasonably good repair, after deducting the estimated expense of removing the nuisance or putting them into good sanitary condition or repair; (*c*) that the house or premises are unfit for, and not reasonably capable of being made fit for, human habitation, *the compensation shall simply be the value of the land and of the building materials thereon.*

PART II.—UNHEALTHY DWELLING-HOUSES.

This part of the Act rests mainly on the Artisans' Dwellings Act of 1868 (Torrens's Act), and the Acts of the two previous years, and deals with (*a*) "Buildings unfit for

human habitation", and (*b*) "Obstructive Buildings". The procedure, however, provided by the Act is new and may be summarised as follows:—This portion of the Act is set in motion by the Medical Officer of Health, whose duty it shall be to represent to the Local Authority of his District, any dwelling-house which appears to him to be in a state so dangerous or injurious to health as to be unfit for human habitation. He can be set in motion by a complaint of four or more householders preferred to him in writing. Failure in dealing with the matter may be followed by a petition of the householders who signed the original complaint to the Local Government Board for an inquiry, and the Board may, after such inquiry, order the Local Authority to proceed under this part of the Act. These powers are materially strengthened by Sect. 32. (1.) It shall be the duty of every Local Authority to cause to be made from time to time inspection of their District, with a view to ascertain whether any dwelling-house therein is in a state so dangerous or injurious to health as to be unfit for human habitation, and if, on the representation of the Medical Officer, or of any Officer of such Authority, or information given, any dwelling-house appears to them to be in such state, to forthwith take proceedings against the owner or occupier for closing the dwelling-house. The Magistrates may make a closing order and impose a penalty not exceeding twenty pounds; and after that order, the Local Authority may, if they think fit, resolve on the demolition of the building; and, after hearing the owner upon such resolution, they may (unless the owner undertakes to put the building in a state fit for habitation) order the demolition, and themselves demolish the building accordingly, if the owner does not do so within three months, and defray their expenses by sale of the materials. An appeal lies from the Local Authority to the Quarter Sessions.

Obstructive Buildings.

An obstructive building, although not in itself unfit for human habitation, is so situated as to stop ventilation, or otherwise makes or conduces to make such other building to be in a condition unfit for human habitation or dangerous or injurious to health, or to prevent proper measures from being carried into effect for remedying any nuisance injurious to health or other evils complained of in respect of such other buildings; and may be pulled down on the representation of the Medical Officer of Health, or from four or more inhabitant householders of the District. When the building has

been pulled down, the land may be left in the possession of the owner, compensation only being paid for the estimated value of the building, or it may be utilised as an open space, or for street improvement, and not for the erection of any other similarly defective building; but the space may be used for workmen's dwellings, with the consent of the Local Government Board.

Where it would be beneficial to the health of the neighbouring inhabitants, if the area of the demolished dwelling-house were utilised as a highway or open space, or appropriated, sold, or let for the erection of dwellings for the working-classes, or exchanged with other neighbouring land which is more suitable for the erection of such dwellings, and on exchange will be appropriated, sold, or let for such erection; or where it appears that any buildings are dangerous or prejudicial to health, and that the demolition or the reconstruction and the rearrangement of such buildings or some of them is necessary to remedy the said evils, and the area thereof is too small to be dealt with as an unhealthy area under the first part of the Act, the Local Authority shall pass a resolution to the effect, and direct a scheme to be prepared for the improvement of the said area. An order confirming the scheme has next to be obtained from the Local Government Board (which does not, unless appealed against, require the confirmation of Parliament), and which may give powers of compulsory purchase. The Board may require provision to be made for displaced persons. The arbitrator is to take into consideration, in settling his award, any increased value which, in his opinion, may be given to other dwelling-houses of the same owner, by the alteration or demolition of any building. There is no appeal from the arbitrator's decision.

Part III.—Working-Class Lodging-Houses.

Where the Labouring Class Lodging-Houses Acts of 1851 and 1855 have been adopted, this part of the Act is applied, and has the force of law: in other cases it is permissive. The expression "lodging-houses for the working-classes" includes separate houses and cottages for the working-classes, whether containing one or several tenements, and the purposes of this part of the Act include the provision of such houses and cottages. It may include a garden of not more than half-an-acre, provided that the estimated annual value of such garden shall not exceed three pounds. Land may be acquired for the purposes of the Act by the Public Health Act of 1875. The Local Authority may erect lodging-houses

on any land so appropriated, or they may purchase or lease lodging-houses already erected or to be erected. The management of such lodging-houses is vested in the Local Authority, who may fix charges for occupation thereof, and make bye-laws for the regulation of the same. When lodging-houses are found to be too expensive or unnecessary, they may be sold after they have been established seven years.

What has hitherto stood in the way of the more frequent application of the Artisans' Dwelling Act (Cross's) has been: (*a*) the great expense incurred thereby; (*b*) a want of due appreciation, on the part of the municipal electors and their representatives, of the vital importance of healthy homes and surroundings for the working-classes; (*c*) the practical retention until lately of the balance of legislative, municipal, and administrative powers in the hands of those directly or indirectly interested in the maintenance of the *status quo*. That statute, and its amending statutes, have now been repealed.

The Housing of the Working Classes Act, 1890, which takes its place, consolidates a good deal, and amends too little. The chief difference between the two statutes is that, in the new one, some of the compensation clauses have been improved. The ten per cent. or more which had by law to be added to the price fixed by the arbitrator, in cases of compulsory purchase, is disallowed. The market value of any house maintained for illegal purposes is to be regulated by such a rental as might be obtained if let for ordinary use. These are valid concessions so far as they go; but they are utterly insufficient. It is true that any house condemned as unfit for human habitation (Part II) may be, after a rather circumlocutionary process, pulled down, and the material sold for what it will bring, to recoup the Local Authority, and the land on which it stood purchased compulsorily in the usual way.

But the cost of clearing away and purchasing under Part I is still exorbitant and unjust. Any given house situated in an area condemned as insanitary is valued without making adequate deductions on account of its unhealthiness. A rent artificially exalted by the necessities of a helpless tenant and the exactions of a rack-renting owner—supported by a law originally framed and enacted by, and for, the benefit mainly of the class to which he belongs, forms the basis of valuation. So invincible have the landlords made themselves in this matter, that the ratepayers as a body have to pay an unreasonably high price for property declared to be practically worthless for the purposes for which it was con-

structed and maintained. It is much to be regretted that, as the law stands, the owners of such insanitary property cannot be compelled to pay something for its improvement. The cost falls upon the already over-burdened ratepayers, and the real delinquents escape "even a fine for this gross neglect of their duties".

It is extremely difficult to understand on what principle of equity or fair-play it can be maintained that property of this kind, which produces a luxuriant harvest for the undertaker, the prison, public-house, and the workhouse, should be altogether paid for by the ratepayers. Something should be done to make the slum owners pay a proportion for the excessive mortality, low standards of vitality, disease, crime, vice, and misery, for which they ought to be held partially responsible.

If the existing law and practice are to be perpetuated, they will have to be met by such a taxation of ground-rents, and of the neighbouring property improved by the operation of the Act—encouraged already to a very slight extent by Part II—and also, if needful, by such a scale of municipal death-duties as will cover a fair and equitable part of the expenditure. It is unreasonable to expect that the occupiers of house property in towns and cities will tolerate much longer the present system of saddling an undue proportion of the cost upon them.

The recent decision of the London County Council to deal radically with "the unhealthy area of Bethnal Green", covering about fifteen acres, at a cost to the ratepayers of £300,000, involving for the first year a charge of £14,000 for interest and sinking fund, is a noble endeavour to give healthy homes to not really a very large number of persons. This is seemingly regarded by the public as a gigantic scheme; yet, compared with what is being grappled with elsewhere, it is relatively a small one! I know a town, about thirty-three times smaller than the metropolis, which has just tackled two "insanitary areas", at an estimated cost of about £55,000; and is ready to do more and more, until every slum and unhealthy house are replaced by wide, well-sewered streets and healthy habitations. To place London, in this respect, on a par with the enterprising borough to which I refer, an expenditure of at least £1,815,000 would be required. A scheme of this magnitude might be fairly designated as gigantic.

In every town of any dimensions there exist unhealthy areas and houses demanding radical improvement, re-arrangement, and reconstruction under this Act, with all practicable

expedition. The important departure just taken by the new municipality of London ought to accentuate the necessity of tapping additional sources of income, so that this method of brightening the life and bettering the health of the people may be widely and speedily extended. To accomplish this object a liberal amendment of the Act will have to be made. With an effective reduction of the cost of compulsory purchase, the discovery and application of additional resources, there is no valid reason why the process now inaugurated in London and elsewhere should not be imitated and pushed forward to even greater conquests in every town in the country, until all unhealthy areas are effectually disposed of. In surveying the question of providing efficient shelter and accommodation for the masses, it should be borne in mind that our real wealth resides in the health of the people.

There is in the decided fall in the average death-rate of the country from 22.42 per thousand in 1866-70 to 17.9 in 1889, and in the corresponding rise in the average duration of life, irrefragable evidence of the improvement of the general health which has chiefly followed the administration of the Public Health Act of 1875, especially in those localities where it has, in recent times, been supplemented by local statutes, conferring extended powers. But, even so assisted, there are limitations to its operation. It can, so aided, and when wisely administered, provide every house with a separate water-supply for domestic use; that water set apart for flushing closets cannot be used for other purposes; that pipes, leading from sinks, duly trapped underneath the same, are made to open through a wall in the open air over a channel leading to a properly trapped gully grating; that pipes of baths are similarly trapped and guided; that all badly placed stack pipes are thoroughly disconnected from soil drains; that properly-ventilated, trapped, and flushed closets are employed; that the soil pipe at or near the point of issue from the closet trap is ventilated by means of a four-inch pipe (outside) leading above the top of the house, well away from chimneys, windows, and skylights; that the soil drain is effectively cut off from the sewer; that all cesspools discovered are closed, and the houses to which they belong are properly connected with the sewer; that houses found to be drained on the joint system are separately drained; that new drains, or old ones renewed, are constructed of the best material, well jointed, laid in cement, and only passed by the surveyor after standing the water test; that, as regards new buildings or old houses rebuilt,

the adjoining sewer is, in suitable cases, ventilated well above roofs, whenever practicable or desirable; that all old wells are closed up; that dust-bins (portable zinc ones are, perhaps, the best) are kept as free as possible from putrid vegetable matter, emptied at least once a week, or oftener, as in the case of hotels and other large establishments.

This Act can give pure water. It can secure the construction of a well-ventilated system of arterial and house drainage. As to what it can do, in conjunction with a Local Improvement Act, Brighton may be cited as an example. The death-rate there was, about twenty years ago, 22·01 per thousand; last year it was 15·04. But, even so supplemented, it fails to deal effectively with badly constructed, dilapidated, and rotten tenements, whose floors and walls are soaked with filth, hemming in and shutting out courts and alleys from proper light and ventilation; standing on ground sodden and saturated with the filth of generations or of centuries, and permeated by a semi-stagnant atmosphere laden with heavy, sickly, and deleterious exhalations. It may partially mitigate or scotch these evils; but it can neither remedy the abominations of a house absolutely unfit for habitation, nor the moral infamy and physical impurity of the slum or "unhealthy area"—the breeding ground and home of infectious disease.

This condition of things can only be eradicated by the root and branch process provided by an improved Housing of the Working Classes Act. The best authorities seem to agree that, by sweeping away unhealthy houses and areas, and replacing them by wide, well sewered streets and good houses everywhere, the death-roll might be reduced to 10 per thousand. But the clearing process will not advance with the desired speed until ways and means have been devised, somewhat in the manner previously mentioned, to alleviate the burden at present thrown upon the ratepayers in carrying out the Act. Seeing the great object to be accomplished, the sooner this relief is secured the better. Such a death-rate effected by a radical reformation of the slums means a higher standard of health and vitality all round, and an immense reduction of sickness, both of a trivial and serious character; it means, further, in all probability, not only the practical stamping out of the endemic infectious diseases, including consumption, but absolute security from the diffusion of epidemic cholera in these islands. This is the ultimate aim of the sanitarian and epidemiologist. High it may be; but is it not well and wise to aim high, especially in such a splendid cause?

The increased powers conferred in Part III of the *Public Health Amendment Act*, 1890, which embodies many advanced provisions contained in local statutes, not included in the Public Health Act of 1875 and its Amending Acts, will, in all places where the new Act is adopted and vigorously applied, contribute materially to this end. Like too much of the sanitary legislation of the last Session, this part of the Act is permissive, and can only be applied after it has been adopted by the Local Authority. But wherever it is adopted and acted upon it must be followed by good results. In illustration of this, some of its more important provisions may be concisely summarised. (1.) Any person interfering with the free flow of sewage, surface or storm water, in any sewer or drain connected with it, after due notice has been served, by the admission of chemical refuse, waste steam, condensing water, heated water, or other liquid of a higher temperature than 110° Fahr., which either alone or with the sewage causes a nuisance, or is dangerous or injurious to health, or is calculated to injure the structure of the sewer, will be liable, under Section 17, to a penalty not exceeding £10, and to a daily penalty not exceeding £5. (2.) Where a system of sewerage is being carried out, an owner or occupier of any premises entitled to have the drainage of the same connected with the public sewer can, under Section 18, by paying the estimated cost in advance, require the Sanitary Authority to make such communication, and also, by agreement, even to make, alter, or enlarge for him any private sewer or drain. (3.) Although by the *Public Health Act* of 1875 the Local Authority may provide and maintain urinals, water closets, earth closets, privies, ashpits, and other similar conveniences for public accommodation, it omits to confer powers to regulate the use of the same. By Sections 11 (3) and 20, the Urban Sanitary Authority may frame regulations for the management of any such "sanitary convenience". They may make bye-laws as to the decent conduct of persons using them. They may let them for a term not exceeding three years, and they may charge fees for the use of water closets. (4.) Penalties may be imposed, by Section 21, on persons injuring or improperly fouling sanitary conveniences used in common by the occupiers of two or more separate dwelling houses or by other persons. (5.) By Section 22, it is enacted that all workshops or manufactories in any district where Part III of the Act has been adopted, *must* be provided with efficient and suitable sanitary conveniences, having regard to the number of persons employed or in attendance

therein. Where persons of both sexes are employed or are in attendance, an adequate number of suitable sanitary conveniences for each sex must be furnished in separate situations. (6.) By Section 23, Urban Sanitary Authorities are enabled to make bye-laws with respect to (*a*) the efficient flushing of water closets, (*b*) the structure of floors, hearths, staircases, and height of rooms to be used for human habitation, (*c*) paving of yards and open spaces in connection with dwelling houses, (*d*) the provision, in laying out new streets, of secondary means of access where necessary for the removal of house refuse and other matters, (*e*) the drainage of houses, water closets, earth closets, privies, ashpits, and cesspools, flushing of water closets, both in new and old buildings. (7.) By Section 23 (4) it is enacted that buildings originally constructed in conformity with the bye-laws in force at the time of their erection cannot be subsequently altered in such a way that if at first so arranged they would have violated the bye-laws. (8.) Section 24 forbids the occupation of any room, whether built before or after the adoption of Part III, "any portion of which extends immediately over any privy (not being a water closet or earth closet), or immediately over any cesspool, midden, or ashpit." Penalties are imposed for infringement of this part of the Act. (9). Section 25 now makes it "unlawful to erect a new building on ground which has been filled up with matter impregnated with fæcal, animal, or vegetable matter, or upon which any such matter has been deposited, unless and until such matter shall have been properly removed, or shall have been rendered or have become innocuous." An offender against this provision is exposed to penalties. (10.) Section 26 authorises the Local Authority to make bye-laws for (*a*) fixing the time for the removal or carriage from within or without, or through the district, through the streets of any fæcal or offensive or noxious matter or liquid, (*b*) providing that a properly constructed and covered "vessel, receptacle, cart, or carriage" is employed, (*c*) cleansing of any place on which any such matter or liquid has been spilt in process of removal. (11.) Section 26 (2) authorises the making of bye-laws requiring occupiers to facilitate the removal of house refuse. (12.) Section 27 provides for the sweeping and cleansing by a Local Authority of courts and passages, not a highway repairable by the burgesses, if not otherwise kept clean and sweet, and for the recovery of the cost from the several occupiers. (13.) Sections 116, 117, 118, and 119 of the Public Health Act of 1875, are by Section 28 of the new statute "extended and made to apply to all articles intended

for the food of man, sold or exposed for sale, or deposited in any place for the purpose of sale or of preparation for sale within the district of any Sanitary Authority." It goes farther—a Justice may condemn any article so intended, exposed, deposited, or prepared, and order it to be destroyed or disposed of, if satisfied, on complaint being made to him, that such article is "diseased, unsound, unwholesome, or unfit for the food of man, although the article may not have been seized or mentioned in 116 of the Act of 1875." (14.) The existing law contains no provision empowering the Local Authority to license slaughter-houses for a limited period only. By Section 29 of the new Act, the licence "shall be in force for such time only, not being less than twelve months, as the Urban Sanitary Authority think fit to specify in the licence." Further, any change of occupation of premises, registered or licensed for use as a slaughter-house, must be notified to the Inspector of Nuisances (Sec. 30), whilst the occupier, on being convicted "of selling or exposing for sale, or having in his possession, or on his premises, the carcase of any animal, or any piece of meat or flesh, diseased or unsound, or unwholesome, or unfit for the use of man as food, will be liable to have the licence revoked by the Court." (15.) Although the law requires the keeper of a common lodging-house to give immediate notice to the Medical Officer of Health and to the Relieving Officer of every case of fever or infectious disease occurring in his lodging-house, it fails to exact any penalty for non-compliance, the penalty for which, by Section 32, is now not to exceed 40*s.* and 5*s.* daily.

We are unquestionably on the right path. For, although in the four severe and decimating epidemics of 1831-32, 1849, 1853-54, and in 1866, cholera spread widely in this country, and the travelling form of the scourge in 1871-74, 1884-87, and possibly also the outbreak of the present year in Spain, reached our shores, it failed, on each occasion, to secure a footing and to spread. What is the explanation of this? It can scarcely be argued that the same atmospheric or telluric conditions which in each of these epidemics favoured its migration and diffusion in neighbouring countries in Europe were not experienced in the United Kingdom. The reason is probably to be found in the fact that, since the invasion of England in 1866, instead of making futile attempts to stem its onward progress by barricades. cordons, and quarantine, and so focussing and intensifying the pestilence, we have, on land and water, concentrated our attention on removing from our midst "those insanitary conditions which are essential to its spread, and on increasing the security

afforded by those measures of imperial and local sanitary administration, by which it is sought to diminish sickness and mortality from all preventable diseases, including cholera." (Thorne Thorne.) Indeed England, soon after the epidemics of 1831-32 and 1849, began to recognise the powerlessness of quarantine in checking the advance of the disease, and set to work to frame a series of measures for preventing the spreading of infectious maladies, and the protection of the Public Health. The administration of these, and particularly of the Public Health Act of 1875—aided in many places by local statutes giving increased powers—has now, as we have seen, culminated in making isolated cases of cholera exhaust their force with as little harm as a lighted taper burns out on a marble floor. But although this has happily been so during these three epidemics in Europe, it must not be overlooked that our safety will not be absolutely reliable until all houses unfit for human habitation and all unhealthy areas are everywhere abolished and replaced by wide streets and roomy, bright, and cheerful dwellings for the accommodation of the artisans, workers, and others who may, during the necessary clearance, re-arrangement, and re-construction, have been temporarily displaced.

ON THE CAUSE OF ENTERIC FEVER IN INDIA.

By H. SKEY MUIR, M.D., Brigade-Surg. A.M.S.

(*Read: Dec. 17th*, 1890.)

HAVING observed in the *British Medical Journal* (24th May last) a short abstract of a paper read by Surgeon-Major Pringle at a meeting of the Epidemiological Society, on "Enteric Fever" in India, I venture to submit the following remarks with reference to that portion of the report which refers to Chakrata; as a residence of nearly two years at that station justifies me to a certain extent in considering that I have some acquaintance with its vital statistics.

Surgeon-Major Pringle evidently agrees with the now generally received opinion that impure water is the principal cause of enteric fever in India, and so far I agree with him; but I do not think his information concerning this particular place is very recent, for I have rarely seen so many inaccuracies compressed into two sentences of moderate length.

It is, apparently, the intention to send more troops to Chakrata and the adjacent hill Kailana; for additional huts are in course of construction. And, indeed, a healthier situation can scarcely be selected. I feel constrained to quote some of Dr. Pringle's words, and to comment upon them in defence of the station.

"*Chakrata . . . . cold and bleak.*"

Coldness is a comparative term. Some people feel "chilly" when others complain of "closeness"; and perhaps Dr. Pringle is one of these. Most people who visit Chakrata remark on the *freshness* and *purity* of the air; they say it is not so "stuffy" as Mussoorir (which is forty miles distant by road, less than twenty miles as the crow flies).

But as individual peculiarity, or imagination, is involved in such expressions, we must fall back upon figures. Below, I give the annual mean temperature and rainfall for the last five years. I also show the mean temperature for the nine months, March to November, this being the usual period of occupation. (The present rule is that only a very small number of men—last year it was twenty-six—remain between 15th November and 20th March.) The rainfall averages 29 ins. less than at Mussoorir.

Years.	Max. Temp.			Min. Temp.			Mean Temp.			Rainfall, ins.
	Annl.	Absolute max.	Date of max.	Annl.	Absolute min.	Date of min.	Annl.	Mar. to Nov.	Dec. to Feb.	
1885	62.5	83.3	12 June	48.5	25.1	18 Feb.	54.9	59.5	41.0	80.72
1886	63.4	82.3	13 June	49.4	24.6	3 Feb.	55.9	59.7	43.8	50.80
1887	64.3	84.2	25 May	49.8	23.1	9 Feb.	56.4	60.9	43.0	64.06
1888	64.6	87.3	25 June	49.9	23.1	29 Jan.	56.6	61.0	43.4	71.25
1889	66.4	88.4	3 June	51.3	27.3	2 Feb.	58.7	62.5	47.5	80.42

1889 was an exceptionally hot year, but, if the figures prove anything, I submit that they show Chakrata to be anything but cold and bleak.

"*Magnificent water-supply . . . . had been neglected, and water was carried by hand from a tank below the barracks without pretence at purification, though it received the surface drainage, excreta, and filth from the dwellings above.*"

The "magnificent water-supply" has been taken advantage of for some years. At first, in 1869-70, it was brought from its source, 1,000 feet above the station, in open ducts; which were intended as a makeshift until funds were forthcoming to lay pipes with. The water was so pure that there seemed no reason to alter the arrangement; and I have the testimony of an Engineer officer (who was present when pipes were laid down), to the effect that money was forthcoming only when it was conclusively shown that a large saving could be effected in the distribution of the water by the reduction of a large number of mules employed in carrying it. At this early period (1870) the tanks filled by the open ducts were so situated that the chances of contamination were reduced to a minimum. It, of course, could not be said that disease-germs might not find their way into the open channel, but there were no villages nor habitations of men near any part of the duct, and it was so constructed that the possibility of receiving surface-water was small. Nevertheless, a large filter-tank was used, through which all water passed on its way to the service-tanks.

Pipe-laying, however, was carried out and completed in 1887. Water is received from its source in large tanks, passing through a system of gauze network, which prevents leaves from entering the pipes; and the service-tanks are at various points in the station. No further filtration is necessary; the water is as pure as the most fastidious could desire, for there is no possible source of contamination.

I give an analysis made by Professor Notter, who preceded me in medical charge of the station, during 1887-8.

Dr. Pringle appears to allude to a spring below the south-west side of the parade, where there were four or five tanks which still exist. Water was occasionally drawn from this source, during the hot weather, before the rains set in, but the principal supply was from "Deobund", as above described; and whenever the monsoon burst, the tanks below the parade were at once closed (it being recognised that there was danger of pollution from surface-water).

"*Even an alarming outbreak of enteric fever in* 1888 *had not induced the authorities to avail themselves of the absolutely pure water-supply close at hand, or to collect the rain-water from the iron roofs, which was now wasted.*"

I have shown that the "alarming outbreak" in 1888 came too late to be considered as a salutary warning, for the *pure water* supply had been converted into a still *purer* supply in 1887.

But enteric fever did appear in 1888. There were fourteen cases and four deaths. As the disease had been comparatively unknown in Chakrata previously, I will not split straws with the writer of the paper over his expression, "*alarming outbreak*". I look on the occurrence of enteric fever as a very serious matter, whether the number of cases be few or many. And I concur with Dr. Pringle in the opinion that we must search for its origin usually in the water-supply. But I differ from him *in toto* with regard to his ideas about the supply at Chakrata.

Where, then, are we to look for the source of the enteric poison? Is it in milk? One or two cases that occurred last year seemed to point to the possibility that impure milk might have been the cause. The milk-supply is, however, carefully scrutinised; and, while not losing sight of it, I do not propose to discuss that question, as I think what I still have to say about water is more important.

I have a very strong opinion that the enteric germ is imbibed by troops on the march from the plains to Chakrata; more especially from the railway station of Saharampore upwards for the seventy-seven miles of road which is traversed. The camping-grounds, at some of the halting-places, are made use of by bullock-trains carrying supplies. Water is obtained from wells in some instances, from the adjacent river in others. The wells situated in the camping grounds are made use of freely all through the summer by numerous natives, who, in the manner well known to Anglo-Indians, perform their ablutions so that the water drains off their bodies back whence it came. The river, a picturesque and to all appearance a pellucid stream along whose valley

the road winds for some miles (and which at a place called "Siah" forms the boundary of the camp), passes through two or three large villages, and receives surface-drainage, which must contain contamination, from several others.

Now, if the poison is thus absorbed in the water freely partaken of by the hot and thirsty soldier on the march, what should we expect? We should expect the appearance of enteric fever in certain individuals at a date later than the first day of the march, by the period of "incubation" of the disease. (The period of incubation does not seem to be practically settled by writers on the subject. My own experience in India, agreeing as it does with that of many others, leads me to believe that it is in many instances a month, at least.)

This is exactly what occurred in 1888, 1889, and this year. The cases almost invariably declared themselves within one month of their arrival in the station. The troops here consist of two bodies of men. A whole regiment is quartered upon two hills known as Chakrata proper. On an adjacent hill, called Kailana, which is about two-and-a-half miles from Chakrata, some 750 men (small detachments from several regiments and batteries), known as "details", are located. The "details" generally march up some ten or twelve days later than the regiment. *Accordingly, we find cases of enteric fever continue to show themselves amongst these "details" ten or twelve days after the last case has occurred in the regiment.* Can any fact be more significant?

Towards the end of the abstract of Dr. Pringle's paper he speaks of happy results following his supervision of roadside wells in the Agra district and elsewhere. It is curious that he should have overlooked the possibility that an "outbreak" of enteric fever at Chakrata might be due to the water along the road! Sanitary recommendations, in these days of "cheeseparing", are generally "pigeon-holed" if their adoption involves much expenditure. Strong representations have been made, during the last two or three years, that measures should be taken to prevent the pollution of the wells referred to, but the authorities appear to be somewhat slow in producing any practical results. While, therefore, I defend them from the charge of indifference Dr. Pringle brings against them, with regard to the water-supply of the station, I cannot avoid expressing my firm belief that some twenty or thirty cases of enteric fever will occur in Chakrata every spring, until more care is taken to insure a supply of pure water on the march.

Professor Notter's analysis of the Chakrata water-supply

(1888) :—Chlorine, 1.5843 grs. per gall.; ammonia, *nil*; free albuminoid ammonia, *nil*; nitrous acid, *nil*; nitric acid, a trace; organic matter, none. Very little sediment, chiefly vegetable *débris*. Water very good, free from organic impurity.

The following particulars are in support of my statements. They refer to last year and this year, during which time I have been in medical charge. (Dates of *occurrence* must be considered as the dates of *admission* to hospital; the dates of *attack* should be placed from two to eight days earlier.)

Years.	Troops which marched to Chakrata.		Dates of Occurrence.	No. of Cases.
	Corps.	Date of Arrival.		
1889	4th King's R. Rifles	March 29 & 30	Between April 9 & May 10	7
	Details	April 16 ...	„ „ 16 „ 21	14
1890	2nd Border Regt....	April 2 & 3 ...	Between April 8 & May 4	11
	Details	„ 14 & 15...	„ „ 14 „ 17	9

I regret to say that this year we have had cases of enteric fever since the 17th May, but inquiry has only served to show that the water-supply of Chakrata proper is not in fault. The cases have occurred amongst the "details" only, and I believe the cause has been discovered. It appears that during the hot weather this year, following an unprecedentedly mild winter, there was some fear that the supply of water from Deobund might run short if used by all the troops in the station. Accordingly, the "details" had to draw upon a spring situated upon their own hill (Kailana), and a close inspection of the source revealed the possibility, albeit a small one, of contamination. As soon as the regular rains commenced, the Chakrata supply was again made use of by all. It may be mentioned that the date of attack, in the last case which has occurred, was just within thirty days following the date on which the water from the spring referred to ceased to be used.

IS THE COLONISATION OF TROPICAL AFRICA BY EUROPEANS POSSIBLE?

By Surgeon-General Sir WILLIAM MOORE, K.C.I.E., Q.H.P.

(*Read January* 21*st*, 1891.)

It is now some thirty-six years since I was serving at Neemuch, in Rajpootana, with the 1st Regiment Bombay Light Cavalry Lancers. At that period our distinguished President was also serving in the same province, and engaged in writing his standard works on the *Vital Statistics of Indian Jails*, and on the *Vital Statistics of the Indian Armies*. I must ask you to pardon, even at this early period of my address, a little digression. Our President, in his work, *Vital Statistics of the Indian Armies*, demonstrated that the men of the Anglo-Indian Army *disappeared* at the rate of 69 per 1,000, and no notice was taken. But when, eight years afterwards, the Royal Commission on the Anglo-Indian Army demonstrated the same, Sir Charles Hood, then Secretary of State for India—who ought to have known better—said in the House that the Report of the Commission had disclosed a rate of mortality which, before its publication, no one believed to exist. Our respected President may claim to be one of the original band of sanitary reformers in India, the few survivors of which have the satisfaction of at last seeing most of the recommendations, so long ignored, at last carried out for the prevention of epidemic disease. Well, at the period mentioned above, thirty-six years ago, the late Sir Henry Lawrence, afterwards killed at Lucknow, was the chief official of the province in which your president and myself were serving. Sir Henry Lawrence, who has since been called "the good", was then initiating his philanthropic scheme for the establishing schools and asylums for European soldiers' children on the hill ranges of India. Although I was a young officer at the time, Sir Henry Lawrence asked my views on the subject. He also asked, if I ever had the opportunity, that I would promote his endeavours. This I promised, without any expectation that I should be in a position to redeem the promise. But as events turned out, I was afterwards surgeon and then honorary secretary for a number of years to the

Mount Aboo Lawrence Asylum for European children; and I think I may say this enabled me to fulfil my promise. Sir Henry Lawrence was also very desirous of establishing European colonists, and especially time-served soldiers, on the Indian hill ranges. I was also consulted on this question. The subjects will be referred to again. I merely mention the foregoing facts now because it shows that my attention was early attracted to the questions, and which induced me throughout a long service in the East to give much consideration to the influence of tropical climates on the European, and to the constitutional requirements of the European for tropical climates. It also demonstrates that what I have to say is the outcome of observation and experience. Companies have been formed for the development of Africa. The somewhat questionable blessings of civilisation are to be opened wholesale. Pagans and Mahomedans are to be converted to Christianity. Slavery is to be abolished. All this is to be accomplished without the introduction of spirituous liquors, opium, or firearms, by European settlements. Let us hope such anticipations may be realised. There is, however, I think, a doubt about it. It appeared to me that the subject is one worthy of discussion by this Society; for if an expression of adverse opinion by this Society goes abroad it may be the means of preventing much disappointment; whereas, should the Society express a favourable view it would certainly strengthen the resolution of those who essay to brave the African climate.

Africa has been pictured as possessing the capabilities of a second India. The old "East India Company of Merchants trading to ye East" doubtless had a profitable business. But in Hindustan there was already a civilisation antecedent to our own. And although Indian civilisation had lagged, there was still a wealthy section of the people ready to accept western merchandise. This is not the case in Africa; the taste is yet to be created, and the wealth has to be extracted from the earth, for it does not exist in the pockets (or whatever substitutes may be used for such conveniences) of the African people. There were no cannibals of the Congo, as depicted by Mr. Ward, in India, or dwarfs with poisoned arrows, as described by Stanley. It has latterly been said by Mr. Mackenzie, of the British East Africa Company, that the time has arrived for English help to take a more effective form than mere sympathy and moral support. He pleads for the adoption of the same methods which had to be originally employed in India; to furnish the country with a railway system, that is, a Government guarantee of interest on the

capital expended. But, as the *Globe* observed, "To construct a line from Mombassa into the interior of Africa would necessarily involve very heavy expenditure, and although profitable traffic might gradually grow up, it may be safely predicted that the line would not do more than pay its working expenses for many years. In the case of India, the traffic was already in existence, and the two cases do not, therefore, run on all fours. Besides, the guaranteed interest in that instance was paid by the people of India themselves, not by the British taxpayer, and we very much doubt whether the latter would care to have his burdens increased for the development of Africa."

Now the civilisation of Africa is to be accomplished by the presence of Europeans in the dark continent, or, as some do not hesitate to advance, by the colonisation of Africa by Europeans. But unexpected contingencies are apt to interfere with the most carefully laid plans. The contingency, which certainly should not have been unexpected in this instance, but which nevertheless does not seem to have commanded attention, is the probability that Europeans will not be able to live and flourish in an African climate. It is true that we find mankind fitted for many climates, to lofty mountains and to tropical swamps, to temperate, frigid, and torrid zones. It is also true that Europeans have endured winter in the Arctic region with a temperature from 40 deg. to 50 deg. below zero; and have lived on the banks of the Senegal in the hot season, where the temperature ranges from 120 deg. to 130 deg. But as fitness for one set of conditions of climate, so usually unfitness for opposite conditions. The Anglo-Saxon race, excepting probably the Jews, are perhaps better fitted to brave extremes of climate than any other section of mankind. But there is a limit to the endurance of even the Anglo-Saxon. Because man possesses the faculty of adapting himself to climates different from that in which he was born, it does not follow that this power is without limit. A climate favourable to the integrity of one set of organs is often hurtful to another set. Thus, a climate favourable for the pulmonary organs may promote hepatic disease. And immunity from rheumatism may be purchased by liability to fevers. Bonden, long since, truthfully asserted that the mortality of Europeans in hot climates is not lessened by lengthened residence, but the reverse. Our experience in India demonstrates this. If colonisation in India, as America and Australia have been colonised, were possible, some instances of the kind would have occurred. But the truth is the European race dies out in India. It

was lately observed, in an annotation in the *British Medical Journal*, headed "Englishmen in Tropical Africa", that in some parts of India, not only in the hills but elsewhere, as at Poonah, the rearing of children of pure European parentage to a normal and healthy maturity is possible, as a result of improved habits of life and sanitation. But, while admitting this may exceptionally be the case, my experience in India leads me to say that a first child is often sacrificed to ignorance and inexperience. The next one or two may perhaps do well, the remainder are weakly and die of diarrhœa, dysentery, convulsions, or some form of atrophy. And what about the children of those who survive? Of the numerous pensioners who have settled at or near the principal Indian civil and military stations none have been colonists. There is not a great-grandchild of these people of pure European descent. It is the same with the descendants of the Portuguese, once the dominant power in India, at least on the sea-coasts. No descendant of these Portuguese can be found without admixture of native blood. And the Portuguese being derived from a southern European country, might be expected to be better fitted for the tropics than Anglo-Saxons from a country far north. An infusion of native blood is, however, an essential to the continuance of an European family in the tropics; and the barrier once broken down, unless special care is taken, the remoter descendants partake more and more of the mental and physical characteristics of the native. When two distinct races are in contact, and there is mixture of blood, the tendency is to revert to the character of that race which is either superior in numbers, or conforms best to environment, especially if, as is the case with the Eurasians of India, they are looked down upon by both Europeans and Indians, and are neglected and uncared for by the State. But my opinion of the half-castes, or Eurasians, of India is much more favourable than is usually entertained; for I have met some very able men and beautiful women amongst them. I think that the Eurasian, if taken care of, would be better fitted for the climate than the European, and, in some respects, better fitted than the pure Indian. And I also opine that eventually the ever increasing class of Eurasians will be a power in the State, and perhaps our principal strength in India. In an article lately published in the *Asiatic Review*, headed "The Falling Rupee", I mentioned that Anglo-Indians were no longer at a premium in the matrimonial market; that several marriages had recently occurred between Europeans and natives; that the necessity of economy would probably increase the num-

ber; and that I would like to see the connection between the two races legitimate instead of illegitimate, for then the children would be taken care of, and instead of evidencing the vices of both races would combine the virtues. I also ventured to institute a mild comparison between the Indian girl and the European girl of the period. But a reviewer said the author had done for himself in every drawing-room in London; and sure enough I have often since been attacked on the subject, especially by matrons with daughters to dispose of. But they were somewhat like the dog in possession, for while not thinking an ordinary Anglo-Indian good enough, they grudged him to an Indian girl. The superiority of mixed races has frequently been advanced, and I imagine something of the belief must have been in Tennyson's mind when he wrote the lines:

> "I will take some savage woman,
> She shall rear my dusky race;
> Iron-jointed, supple-sinew'd, they
> Shall dive and they shall run,
> Catch the wild goat by the hair,
> And hurl their lances in the sun,
> Whistle back the parrots' call, and
> Leap the rainbows of the brooks,
> Not with blinded eye-sight pouring
> Over miserable books."

As there is certain to be a mixed race in Africa, I think those in authority out there, should, from the first, foster this race. They should take warning from the condition of mixed races in other tropical countries, and early devise measures for elevating, rather than depressing the migogen. But can we count upon an enduring mixed race in Africa? It has yet to be proved whether an admixture of Negro and European blood will prove as satisfactory as the East Indian Eurasian. A writer in the *Times* of January 8th, 1891, on the "Negro Question in America", affirms that the mulatto born of the Anglo-Saxon and Negro, rarely attains the age of fifty, and is particularly subject to certain diseases. He affirms that nature refuses to perpetuate this race of hybrids. This inappropriate hybridism is checked by the law of sterility. It is not the same with Indians and Europeans, who to some extent are radically related; that is, derived from a similar stock of ancient times. Many years ago it was the opinion of some of the ablest men in India, including, as before referred to, the late Sir Henry Lawrence, that it would be both politic and practicable to establish colonies on the slopes of the Himalayas. After the Indian mutinies the subject was revived and much discussed. I then wrote

in my book, entitled *Health in the Tropics, or Sanitary Art Applied to Europeans in India,* that "the policy was unquestionable, for European colonists would prove a source of strength in a probable hour of need", and who after the experience of 1857 (then fresh in recollection) "shall say when that hour may not be at hand?" But as regards practicability I observed, "that it is questionable if suitable and remunerative employment could be found for settlers on Indian mountain ranges . . . . much of the hill-side is rock, or too steep for even terrace cultivation . . . . that cultivation of the hill-side under a tropical sun is not work fitted for a European . . . . and that the valleys in the hills are so deadly malarious, that the pioneers of colonisation would be destroyed." As a matter of fact, during the past thirty years, no colonisation has taken place. Buckle long since designated the condition of the people of hot moist climates, as a deteriorated state to which they are doomed by physical laws, impossible for them to resist. And I say that a degree of anæmia must be considered physiological in most hot climates. Anæmia is to be regarded as a malady, quite as important in its connection with, and influence on, other maladies. For there is no disease from which persons suffer in the tropics which is not invited by anæmia, and which is not aggravated, if present, by anæmia. A hot climate sets its seal upon all living therein. Thus the value of native life in the East is considerably less than the value of life in Europe.

Although some doubt has been recently cast on the Aryans being the first invaders of Hindustan from the north, there is no doubt that there was an invasion of Aryan tribes, some making their way into southern Europe, others penetrating into India. From the Shastras we know that these invaders were a white people. And they are generally regarded as the progenitors of the modern Hindoo. It was presumed that Aryan blood is as extended as Aryan speech throughout Hindustan. But Mr. Isaac Taylor, in his recent work, *The Origin of the Aryans,* points out, what is quite correct, that the actual descendents of the Aryans are rare. They are represented by some Rajpoot families and by certain Brahmins of Benares, and other cities on the Ganges, all being people, chiefs or priests, who do no manual labour for a livelihood. Of the 140 millions of Hindustan, it is believed there are not 10,000 pure Aryans. Neither would there have been so many had the early Aryans been suddenly translated to India *viâ* the Suez Canal instead of invading the country from far north gradually during centuries, moving

a few miles every generation, with their flocks and herds and sacred Shastras, and continually amalgamating with the so-called aborigines (who also probably originally came from the north), as the frequent terms now in use, Bramin-Rajpoot, Bheel Rajpoot, Minar-Rajpoot and Ghond-Rajpoot testify. The fact is, for the white man and his offspring there is no acclimatisation in India. It is the same in other tropical countries. The Dutch have failed to naturalise themselves in Java and Sumatra, and they have left no descendants in Ceylon. In Egypt no foreign race has ever maintained itself. The Egyptian fellah still exhibits the precise type seen upon the monuments. Justin Winsor, in his large *History of America*, mentions only two States where the winter wants the severity which characterises the northern parts, and the considerable elevation of the districts relieves them from the dangers of fevers, and "which districts have maintained the original energy of the race that founded its colonies." Next to deserts and snowy mountains, swamps are the greatest barriers to the movements of men. In America there are comparatively few swamps and swamp forests. Peru, which is situate between the equator and the tropic of Capricorn, and by consequence of elevated plateaux affords different climates, is still the country of the Yncas: and this although for many years past there has been an annual influx of Spaniards. Mexico with its *tierras calientes* and *tierras frias* may also be referred to. Mexico has been invaded by Europeans since early in the 16th century, and was formerly called New Spain. The French have also dominated there. But no European nation has there established itself. Yellow fever and black vomit is endemic on the *tierras calientes*, and consumption on the *tierras frias*. A late writer, Mrs. Gooch, in her book, *Face to Face with the Mexicans*, referring to the custom of covering the mouth, says, "Ask a Mexican why they draw the blanket over the mouth? and you will at once be answered, '*Por el aire!*' ('On account of the air!')" Dr. Ahearne, President of the North Queensland Medical Society, has recently made some observations tending to the conclusion that children born in the north or hot part of Australia exhibit a racial type different from children born in the southern or cooler part. They are pallid, thin, and nervous. And he thinks emigration to the north of Australia should be systematically encouraged, as the best means of arresting the deterioration of the British type.

It may be stated as an axiom that northern races die out if transplanted to the south and that southern races become extinct in the north. As a rule, fair races succeed only in

temperate zones, and the dark races only in tropical or semi-tropical lands. Even in Algeria, it is authoritatively stated that French from the north of France fail to become acclimatised, while those from the south of France succeed better. It is also authoritatively stated that, of negroes in the northern States of America, one out of every four is born deaf and dumb, blind, idiotic, or insane, while the majority are sickly and especially liable to pulmonary affections.

Time will not permit of a full demonstration how a tropical climate is injurious to Europeans, but I will briefly mention the principal influences. The first is a depressant effect on the nervous system, a condition with which we are familiar in extraordinary hot weather even in this country. Next the quality of the blood is deteriorated. Air rarified and expanded by heat contains less oxygen in a given bulk, consequently less oxygen is inspired. Owing to the depressant effect there is less disposition to exercise, so that respiration and consequent increase of O. is not thus accelerated. There is also another cause of diminution of O. Years back, Dr. Forbes Watson, of the Bombay Army, showed that the red blood corpuscles of Europeans in India, especially during the seasons of rain-moisture and heat, become smaller, lose their smooth appearance, and are studded with granules of a refractory white, probably fatty nature. Those who are familiar with the pallid, flabby appearance which most Europeans assume in hot, moist Bombay, and on the Malabar coast generally, especially during the rainy seasons, will appreciate the significance of Dr. Forbes Watson's researches. Watson, however, being, I believe, obliged to leave India from illness, did not further describe what it appears not improbable was, twenty-five years afterwards or more, brought forward as the *bacillus malariæ*. Whether this is the case or not, it may with every degree of probability be assumed that the degenerate corpuscles fail in their function of conveying oxygen. Although the diminution of inspired O. from all causes may be slight, it is *continuous*. The higher the temperature the less the exercise, and the more the blood degeneration, the less O. inspired, and the less CO_2 expired, to the extent, as has been calculated, of 25 p.c. Owing to the heat, and the consequent increase of sensible and insensible perspiration, there is a smaller quantity of urine, so that there may not be sufficient fluid to hold in solution all the effete matter which should be passed in the urine. All this leads to accumulation of effete matter in the system, which may be relieved by an attack of boils, or even by prickly heat. In my recently published book, *The Constitutional Requirements for Tropical*

Climates, I said this, but a reviewer in the *Practitioner* objected that in these days of exact science the statement requires justification. Well! we know that the conditions described above do occur, and we know that comparative new comers into tropical climates do very often suffer from boils, the more certainly if they continue the habits of eating and drinking they were accustomed to. After which, for a period, they have better health. More, if there is no attack of boils there will be diarrhœa, caused most frequently by intermittant increase of bile, and the liver is often increased in size. It would appear that this organ compensates for the insufficiency of respiration, and for the diminished urine failing to carry away all the effete matter which should be so thrown out of the system. With these changes the muscular force is no longer vigorous. Neither must it be forgotten that the hotter the climate the more sensitive the skin, and consequently there is extreme liability to chill from slight vicissitudes of temperature. Now, as we all know, many people are rendered irritable by extraordinary heat, even in temperate climates. But when the blood becomes impoverished, altered in character, surcharged with effete material, the brain becomes more irritable. "The reason calm, the temperate will, endurance, foresight, strength, and skill," are all qualifications for a successful colonist; but the changes produced by climate reduce such qualifications to a minimum.

The profound modification in the European system produced by prolonged heat I regard as hereditarily transmissible. It is a fact that the children of parents who have suffered much from so-called malarious diseases are more prone to such maladies than the children of healthy parents. They are sometimes born with a large spleen, and they may suffer from ague before they are born.

The longer Europeans remain in a tropical country the more they feel the effects of the climate. Even domestic animals of colder climates, sheep, horses, dogs, degenerate and often die in the tropics. Mr. Johnson mentions that dogs imported from Europe rapidly degenerate in Africa. This is also the case in tropical climates with many imported vegetables. It is true that Englishmen have exceptionally attained a ripe old age even in India. Men with a larger amount of strength and vital force than others, and possessing the suitable temperament and constitution—particularly if they take care of themselves—can bear exposure and the effects of tropical heat much longer than those not so gifted. But, as a rule, the deteriorating process, if slow, is nevertheless certain; and there is no doubt that this degeneration caused

by tropical climates is in some degree hereditary. Africa lies almost entirely in the torrid zone, and is the hottest continent of all. The west coast of equatorial Africa is, as everyone knows, perhaps the most unhealthy climate for Europeans in the world. The shores of the Red Sea, as I personally know, are also most inimical to the European constitution. Then, rounding Cape Gardefui, there are about eighteen degrees of coast, nominally under British protectorate, and inhabited by Samali and Galla tribes, and which stretches several degrees south of the equator. Then we have the "British Imperial East African Company", extending about four-and-a-half degrees of coast. This tract runs in a northwest direction, bounded north by the Tanga River, stretching over the equator, and abutting on the Victoria Nyanza Lake. South is the German sphere of influence, comprising about six-and-a-half degrees of coast, also abutting on the Victoria Nyanza, the Congo State, Lake Tanganika, on Stevenson's road, and on Lake Nyassa.

Next there is the Portuguese Mozambique coast, with the Zambesi river about the centre, extending to nearly 25 deg. south of the equator. Now, it is well known that all these coasts present more or less of a fever-belt of mangrove swamp and low-lying land, extending longer or shorter distances into the interior. There is sufficient evidence to warrant the assertion that the shores of East Africa do not much differ in climate from the shores of Southern India. Reasoning from analogy, it may be confidently stated that such parts of the African continent are altogether unfitted for Europeans. If analogous reasoning is not sufficient, hear what Messrs. White and Felkin in their book, *The Development of Africa*, say of costal Africa: "It is subject to intermittent malarial fever in an extraordinary degree, and the reason is not hidden, the high mean annual temperature, and excessive rainfall and humidity, being the causes. . . . . To turn up the virgin soil is to release the messengers of death. . . . . Few escape fever. Some die after an attack or two; some drag on, the poison degenerating their blood and tissues, for years. . . . . Europeans, by long residence, lose so much of their moral as well as their vital powers that they are only able to combat the subversive elements of life in an ever lessening degree."

But it will be advanced that European occupancy and colonisation is not advocated for the lowlands of Africa, but for the interior and elevated plateaus. Here, it has been stated, that a climate is found suitable to the human race. This, however, may be doubted, until we get as far south as the highlands to the south of the Zambesi in Mashonaland.

Here, Sir John Willoughby says, there is "a white man's land". Even so far from the equator, Sir John Willoughby's enthusiastic report must be accepted with many salt grains.

From what we know of the interior of Africa, there is the Sahara, or Great Desert, to the north-west, and the Soudan to the north-east, while to the south there are arid plains, known as the Kalahari. Each of these are almost rainless tracts, but subject to heavy dews, and, like all sandy wastes, to great diurnal changes of temperature. Between these sand-tracts there are elevated plateaus.

The isothermal lines of mean highest temperature include the centre of Africa, from Nubia to south of Zanzibar on the east coast, and Guinea on the west coast. So far as is known, the mean temperature, for at least 10 deg. north of the equator and 15 deg. south of the equator, is from 80 deg. to 85 deg.; in some localities, according to Stanley, 87 deg. There are also localities where the mean temperature during the two extreme months is 90 deg. One of these districts is to the north of the equator, in the country of the sources of the Nile. A second is the high plateau country, between Lake Tanganika and the Orange River. The only other parts of the world where an annual mean temperature of 85 deg. has been noted are the southern peninsula India, Central Mexico, New Guinea, and the very north of Australia.

North and south of the African zone of highest temperature there are other wide tracts, with a temperature of 80 deg. gradually diminishing to 70 deg. North, this includes the Sahara and Libyan desert regions; south, the Kalahari, stretching from the south-west coast to the Transvaal and Orange Free State. Broadly speaking, it may be stated that in Africa a mean annual temperature from 85 deg. to 70 deg. prevails almost everywhere. Only in the very highest regions do we find a temperature of from 64 deg. to 70 deg. It will, therefore, be seen how relatively few places there are in Africa where a temperature fitted for Europeans can be found.

While thus dwelling on the importance of high mean temperature, I do not lose sight of the fact that places which have the same mean temperature may have different climates. A place having a moderate summer and winter may have the same annual mean temperature as one having a very cold winter and a very hot summer. But, in the tropics, we have a very high mean temperature with little or no cold weather fall, and it is the continuous heat which is so injurious.

Much has been made of the fact that in the interior of Africa the annual range of temperature is small. This is

certainly the case, and is so far advantageous. But Mr. White and Dr. Felkin, authors of *The Development of Africa*, who have investigated this matter, state that there is an unusually large diurnal range. And it may be doubted if this compensates for the entire absence of a cool season. For there is no doubt that the body and the mind receive certain advantages from the cold season of tropical countries if ordinary care is taken to avoid chills.

Although several conditions besides temperature are factors of climate, continued high temperature I take to be the most important of adverse climatological factors. For there is an optimum of heat which is suitable to different forms of life, but if the heat rises above the optimum it is the reverse. And this is especially the case if the heat is combined with humidity. Then we have the acme of a climate inimical to the European constitution.

Nearly the whole of the central belt of the continent is protected by forests supported by a heavy rainfall. There are double rainy seasons within the central belt. And near the centre of the continent the two rainy seasons become so lengthened as almost to merge into one period of rains, extending over about eight months, during which more than 100 inches of rain fall. Mr. White, in his *Development of Africa*, truly observes, that the climate is the more trying on account of the great relative humidity combined with heat, which enables the palm to flourish on the African coasts as far south as Natal. It has been estimated that about half the continent of Africa is composed of desert and steppes; a fourth part is occupied by savannas; a fourth part being fairly fertile land, mostly covered by forests. The soils available for agricultural purposes are those great flats which border the streams, rivers, and lakes. Or, in other words, the districts which are lowest, which are nearest the equator, which are the most malarious, and where turning up the soil would certainly lead to so-called malarious diseases.

It must be recollected that the climate of elevated regions of hot countries is still a tropical climate. There is a vertical or semi-vertical sun over both plains and mountains, and the characteristic tropical seasonal changes are not absent. There is the hot, a modified cold season, according to latitude and elevation, and a wet season, as on the plains, which are not within the rainless zones. The climate of tropical elevated regions is the climate of the lowlands tempered by that diminution of heat caused by rainfall, forests, and elevation, about one degree for every 320 feet. No doubt this reduction of heat is a very great gain, for the hotter the climate, the more the liability to chill and its consequences.

There is a popular idea that a European, in order to enjoy a suitable temperature in a tropical country, has only to ascend a hill. But to enjoy a mean temperature, like that of London, 51°, in any country where the sea-board has a mean temperature of 80°, it would be necessary to ascend 10,000 feet, and there are no plateaus of that height in Africa. Of course there are elevations. There are mountains 12,000 feet high in Abyssinia. Mount Keni, near the equator, is 6,000 feet; Kilimangara is nearly 23,000 feet. The Livingstone Mountains, at the head of Lake Nyassa, are from 3,000 to 6,000 feet high. But the descriptions extant of these mountains do not afford the least promise that they are suitable for settlements. There are no plateaus of sufficient height known. During Stanley's journey he was not more than 2,000 feet high, excepting when on the plateaus of Kavalli and Undussuma, elevations 4,500 feet, where Jephson, Parke, and Stanley were successively prostrated by fever. Elevated localities have their drawbacks, not only because diseases of temperate climates may prevail thereon, but for other reasons. The principal is the diminution of barometric pressure. Many persons on an elevation comparatively small, experience a sense of constriction of the chest, and sometimes of the abdomen. The pulse and respirations are quickened, and the individual feels that he cannot take a long breath. Slight exertion causes fatigue, and insomnia is common. Any pre-existing heart-disturbance becomes dangerous, and there is liability to congestion of internal organs. Some individuals never become acclimatised to mountain air. Then again there is the hill diarrhœa, or the *Diarrhœa alba* of tropical countries, which it may confidently be expected will prevail on elevated regions in Africa. This malady, I believe, depends on torpor of the liver, the result of change to cold air. For I have frequently known change of colour of the motions and diarrhœa occur within a few hours after the ascent of a mountain. In most cases nothing but removal benefits the patient.

I have known these and other effects of elevation forbid residence at Mahableswar, elevation 4,500 feet; at Pachmari, elevation 3,500 feet; at Mount Aboo, elevation 3,500 feet; and at Matheran, elevation 2,200 feet. Neither are tropical elevations free from malarious fevers. Stanley says there is no immunity from fever and ague from 0 to 5,000 elevation. If lands are to be colonised they must be cleared and cultivated. Clearance of forests tends to lessen rainfall, and to allow the earth to absorb heat, and to radiate it rapidly, thus producing greater vicissitudes of temperature, and, as is known

by Indian experience, would render the climate hotter at some periods, and colder at others. Then the maladies of temperate climates would prevail more in the cold seasons, and those of tropical climates in the hot seasons. The question is, Can such a climate be found under a tropical sun as will permit of the European living, flourishing, and reproducing his race, as in Australia or North America or Canada? That a European can live long on the slopes of tropical hills cannot be disputed, for there are hundreds of instances of the kind in India. But such instances are not afforded by persons obliged to labour, as colonists are obliged to labour for their daily bread. If they are engaged in any business the manual work is performed by natives, for Europeans would not long survive outdoor labour on a tropical hill-side, or especially in a tropical hill-valley. Of the African negro Dr. Hans Mayer says, "He will never be taught by good example and fine precepts, as the missionaries seem to believe; he must be trained in the school of hard work, and he must be forced to work, as he cannot be prevailed upon to do so voluntarily." Not satisfactory to the settler, who hopes to employ native labour.

As regards the propagation of the species, it has been noticed that children brought up in the various schools on the Indian hill ranges, although apparently strong and healthy in their youth, in after years show signs of constitutional weakness, and this has been observed to be specially the case with females. It has been shown that European colonisation and propagation have failed in India and in other tropical countries, and there is certainly no reason why, climatic conditions being similar, it can succeed in Africa.

We cannot accept the statements of some travellers who have been a few months or even a few years in Africa, and who mention a climate fitted for Europeans, because, individually, they retained health; for this is no valid reason why the climate would suit colonists. In the absence of climatological statistics and experience, we must be more guided by what we know of other hot countries. But, as a matter of fact, the great majority of travellers in Africa do not write favourably of the climate.

I now mention what has been said of the African climate by some recent writers. One remarks that: "The interior may be generally described as a fertile region, well suited to the habitation of men, and apparently possessing a climate not unfavourable to life, either for the natives or Europeans who know how to take care of themselves." Another writer mentions that "The climate is not un-

favourable, if care be taken to avoid alcoholic liquors, to lead a regular, careful, and quiet life, and to take plenty of exercise." Mr. White says: "Taken as a whole, it seems evident that Africa is by far the least habitable for Europeans of any of the continents, and generally speaking it is malarial fevers that render it so unfitted for Europeans."

Even Mr. Johnson, in his article in the *Fortnightly Review*, for December last (while expatiating, among other things, on the number of pianos and harmoniums we shall export to an ever-widening circle of custom in Africa), could only say of the climate that when Africa is brought under the same conditions of civilisation and comfort that characterise India, the climate would prove no more unhealthy or fatal to Europeans than India is. Mr. Salter Price has recently published a book, *My Third Campaign in Africa*, where Mr. Price went in 1888 for a few months to visit the stations of the Church Missionary Society, having their headquarters at Frere Town, close to Mombassa. Every few pages there is a mention of some one being sick. Thus Dr. Ardagh, who was living at Mombassa, was "suffering from fever, dysentery, and diarrhœa. . . . . England is pretty well again just now, but has a washed-out look. . . . . Norris has been laid up for the last six weeks. . . . . The journey from Shemba was too much for Burgess, he has been in bed ever since. . . . . On the first day arranged for my interview with the Sultan I was down with fever." After many similar records, it is not surprising to find such remarks as the following: "I was very well my first month, but since the climate has got hold of me, and I am as other men. . . . . It is very hard for an Englishman to live here. . . . Verily death seems very near to us all."

Dr. Hans Meyer, in his recently published book, *The First Ascent of Kilimangara*, says: "The German sphere of interest is a tropical region with definite alternating dry and wet seasons. . . . . In the dry season a high temperature during the day is followed by a low temperature during the night. . . . . The greater part, not only of the German sphere of interest, but of all equatorial Africa, is a sterile, thinly populated wilderness. . . . ." And Hans Meyer quotes Wissman, who said: "One-fifth of German East Africa is good land, the rest is a barren waste. . . . . But the greater part of East Africa is not only sterile, it is unhealthy. Fever is everywhere common, and the plateaus and the habitable mountain regions do not escape. . . . . Those travellers suffer least who are constantly on the move, and who do not stay long in one place, but not one in a thousand escapes

scot-free. If he gets off easily with fever, he is all the more likely to fall a victim to dysentery. The coolness of the nights consequent on the cloudless atmosphere is hailed by Europeans as a welcome relief after the heat of the day; but the abrupt changes of the temperature interfere with the action of the skin and other organs, causing rheumatism and generally diminished power of the organism to resist disease. In East Africa it is impossible for Europeans to live any length of time. . . . . All apparently successful examples of acclimatisation have been short-lived, none ever extending beyond a single generation. . . . . Look where you will throughout the central regions of the Dark Continent, at the trading stations on the Congo, or the mission stations on Lake Nyassa and the Victoria Nyanza, all alike breathe the air of a churchyard, all alike wear the Hippocratic face."

The *Times* correspondent, from even so far south as Mashonaland (*vide Times*, December 4th last), says of the climate there: "As far as we can judge, it is adapted for Europeans," because, "at times a south-east breeze tempers to a degree of actual coolness the rays of a tropical sun." This, I take it, means that there are great vicissitudes of climate, which, in the tropics especially, is destructive.

Again, Laing, in his *Problems of the Future*: "The highlands of Central Africa might probably support a white population."

Mr. Maund has recently (in January) read a paper before the London Geographical Society, "On Matabele and Mashona Land," situated between 16 and 22 degrees south latitude, where he says white children can be reared. "But during and just after the rains", he says, "one must be careful, as in all tropical climates. But with proper precautions, dwellings placed high and above exhalations from the marshes left by the subsiding rivers, above all, judicious abstinence from alcoholic drinks, the new-comer and family will be healthy." Mr. Philips, at the debate following, said he had been there for twenty-six years, outliving all Englishmen who were with him. Even so far from the equator as Durban, in Natal, Mr. Finch, in his *South Africa and Back* says: "There is a maximum mean temperature of 78 degrees and a minimum of 37; moisture, 74 per cent." At Johannesberg, in consequence of the intense heat, he states he felt "all to pieces", and as if "his back-bone were broken".

We are certainly not very familiar with the diseases of Africa, but it may be confidently advanced that they do not materially differ from those met with in other tropical countries.

Asiatic cholera has been known to occur on the Red Sea coast, and as far south on the coasts as Port Natal.

Beri-Beri has occurred on the east and west coasts near the equator.

Dengue has occurred on most of the coast.

Endemic hæmaturia has occurred in Egypt, and on the southern part of the east coast.

Elephantiasis has been observed on the Guinea coast, on the shores of the Red Sea, on the Mozambique, and about the interior lakes.

The filaria sanguinis hominis has been found on the west coast below the equator, and on the east coast opposite Madagascar.

Guinea-worm is found on the gold coast from Mabia to the lakes, and is prevalent about the sources of the Nile, and according to Stanley, in the forests.

Influenza has been recently reported about the lakes.

Leprosy has been seen on the coast of Guinea, in Egypt, Nubia, and about the equatorial lakes.

Malarious fevers extend from the Red Sea to Natal. I find it noted that the natives, when migrating from one district to another, are subject to the malaria of strange localities, though proof against the malaria of their own. I have often known natives of India get sick when removed from their native districts, but have generally attributed it to other causes than malaria.

Negro lethargy is seen on both coasts, but is most common on the Congo. This curious malady has not yet attacked Europeans. It is characterised by unconquerable propensity to sleep, alternating with maniacal conditions, and terminating in coma. Quite recently Manson has found blood filaria in connection with negro lethargy.

Oriental boil, with which we are familiar at Aden, is seen on the shores of the Red Sea, and East Coast of Africa and Zanzibar.

Plague has prevailed in Egypt and along the shores of the Mediterranean and Red Sea, also in Morocco, Algiers, and Tunis.

Scurvy is known from the Red Sea shores to far south of the equator.

Small-pox is also mentioned as prevalent by Stanley.

Tænia prevails from the Mediterranean to the Cape of Good Hope.

Tropical dysentery, like malarious fever, prevails throughout.

Tropical liver-abscess has been specially noted on the

coast of Guinea, in Egypt and Nubia, and on the east coast opposite Madagascar.

Yaws prevails extensively in Western Africa, twenty degrees above and below the equator; on the shores of the Red Sea, on Madagascar, and on the opposite coasts.

Yellow fever is known on the western equatorial coast.

Ulcers are mentioned in Stanley's book, ten times over, as prevailing.

Chest complaints and rheumatism are also mentioned by Stanley.

This is a formidable list for the European settler to contend with. If anyone doubts the impracticability of Europeans colonising tropical climates, I would say go to a tropical climate and study the question. See the European, pale and pallid, struggling for existence, the prey of anæmia, fever, liver-disease, diarrhœa, and dysentery; unequal to all labour, wasted, worn, and finally perishing; and, without influx of new blood, becoming extinct, and leaving children unable to propagate the race.

To summarise, it may be stated that the tropical climate induces chiefly infant mortality, sterility, and anæmia, the latter aggravating and rendering recovery from ordinary ailments more uncertain. Regarding sterility, it may be mentioned that, long ago, Dr. Cutcliff and others in India attributed the sterility of women and the impotency of men, in certain districts of Bengal, to the prevailing malaria, which had become increased by profuse irrigation without drainage. This raised the height of the water in the wells, rendered dry ground sodden, damped the houses of the people, and caused a serious degeneration of the physique of the inhabitants.

In conditions altogether alike, by the nature of constitution, one man incurs more danger than another. This leads me to remark that, if persons will go to Africa, only those should venture who are fitted for a tropical climate. In an article recently published in the *Asiatic Review*, I observed: "When those chronic perplexities children—like accommodation bills—mature unawares, and arrive at the bumptious age, what to do with sons and daughters becomes a burning question. We have probably worked hard for our children, but we now realise that it is worry, not work, which kills men. Therefore the majority are glad when they find some berth for a youth, or some one disinterested enough to take upon himself the charge of another man's daughter. When having, by dint of persistence, secured an appointment abroad for a son, or married a daughter to some one

going abroad, *mater* and *pater familias* opine they have done a good stroke of business. And, having supplied the voyagers with the hundred-and-one unnecessary articles advised by outfitters, and having given the young couple a blessing, *mater* and *pater* sleep the sleep of the just, in the firm belief that, like true-born Britons, they have done their duty. But sending off belongings to the tropics in a haphazard way, like most infatuations, often ends in bitterness and disappointment. Everybody is certainly not fitted for tropical life, and as certainly a great number are totally unfitted. However fond of his children *paterfamilias* may be, it is to be presumed that he does not desire to see his son or his daughter back from the tropics this time next year, if they reappear as confirmed or even temporary invalids. Yet this is what often happens." In my book recently published, *The Constitutional Requirements for Tropical Climates,* I have noted the principal depressing influences of a tropical climate; as heat, liability to chill, prevalence of a scorbutic taint, so-called malaria, want of sleep, and the frequency of a syphilitic taint. And I have noted the following as contra-indicating tropical life: youth and advanced age, hereditary tendency to certain diseases, certain diseases already suffered from, habitual sleeplessness, various peculiar idiosyncrasies, certain temperaments, certain maladies incidental to the female sex. I do not imply that attention to these points would result in sending out a race of colonists. But it would certainly tend to lessen that sickness and mortality which tropical climates now exact from Europeans who venture there. As I mentioned before, I think that an opinion from this Society as regards the possibility or otherwise of Europeans settling in tropical and semi-tropical Africa would be seasonable at the present juncture. For, if adverse, it would probably prevent much future disappointment, and, if favourable, it would materially strengthen the resolution of those essaying the experiment.

THE RELATIONS OF SAPROPHYTIC TO PARASITIC MICRO-ORGANISMS.

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(*Read: February 18th*, 1891.)

MICRO-ORGANISMS, microbes, microzymes, bacteria, or germs, are more or less synonymous terms applied to the low vegetable forms of life—the fungi—which are destitute of chlorophyll and consist of protoplasm enclosed in a cellulose membrane. They multiply by division of their substance, and are, therefore, often termed *Schizomycetes* or *Fission-fungi*. The common property of the majority of these fission-fungi or microbes is that of splitting up organic materials or albumens, under suitable conditions of temperature, moisture, and atmosphere, into alkaloidal and other poisonous or non-poisonous bases and ferments.

The *saprophytes* are those microbes which live only upon dead organic matter—that is to say, those which possess the property of manufacturing organic bases from dead albumens only. To this class belong the bacteria, bacilli, micrococci, spirilla, and vibrios, which exist in such enormous numbers in the air, water, and soil in every region and in every climate, and to which, in conjunction with the yeast and mould fungi, the fermentative and putrefactive changes, to which all dead organised structures are more or less liable, have been ascribed.

The *parasites* are those which prey upon living hosts, vegetable or animal, and which, therefore, in the case of animals, can split up the living albumens at the temperature of the body into organic bases and ferments. Parasites which under no known conditions attack the dead albumens are termed "obligate" parasites, whilst those which are capable of splitting up both dead and living organic materials are termed "facultative" parasites. It should be remarked that the capacity for cultivation in non-living culture liquids, such as peptone-gelatine, agar-agar, blood serum, broth, etc., which is characteristic of many of the

parasitic microbes associated with animal diseases, is evidence of saprophytism, and these microbes should, therefore, be classed as facultative parasites. Of these facultative parasites much has already been learned by reason of this very ability to cultivate them outside the body, and of subsequently ascertaining the pathogenic properties of a perfectly pure culture by inoculation into the bodies of animals. Of the obligate parasites we can assume the existence, but practically know nothing.

It is now a fair assumption, in the light of our acquired knowledge of the ultimate causes of allied communicable diseases, that such diseases as rabies, vaccinia, small-pox, measles, mumps, chicken-pox, whooping-cough, typhus, influenza, relapsing fever, dengue, etc., are the results of the action of specific microbes upon the human body, and as these microbes have not yet been known to exist or cultivated experimentally outside the human body, they may be classed provisionally as "obligate" parasites. Of this class it may be said that further research with improved methods of cultivation will probably transfer many of its members to the class of facultative parasites. The division is, therefore, of a provisional nature only.

Another subdivision of parasitic microbes has been suggested, namely, into (1) non-infectious, but toxic organisms; (2) true infectious organisms. The toxic organisms are held to constitute a link, as it were, between the true infectious and the exclusively saprophytic microbes. They affect the body when introduced within it, only through the poisonous products—ptomaines, leucomaines, albumoses, ferments—which result from their action on the living albumens, and do not cause a general infection of the system. Definite knowledge has still to be obtained of the exact intra-corporeal action of many of the pathogenic microbes with which we are acquainted; but it seems certain that the diphtheria bacillus, according to the researches of Loefller, Roux, and Yersin, is an example of a toxic organism, which produces soluble poisons at its seat of action in the mucous membrane of the throat or mouth, the symptoms of the disease being due to these chemical poisons being taken into the circulation, whilst the bacillus itself is not found anywhere in the body except in the affected mucous membrane. This is true of the disease as it manifests itself in man, cats, pigeons, fowls, etc., but does not apply to bovine diphtheria, where, according to Klein, the bacillus invades the system of the cow, and is found after death in affected organs remote from the point of inoculation. The bacillus of tetanus is also pro-

bably another example of a purely toxic organism, the symptoms of the disease being due to the absorption of an alkaloid—tetanin—produced by the microbe at the seat of inoculation. We might, perhaps, also provisionally classify the pyogenic microbes which are casually related to such diseases as erysipelas, pyæmia, septicæmia, puerperal fever, traumatic gangrene, etc., as examples of toxic organisms, and with still less certainty the microbes of yellow fever, cholera, enteric fever, dysentery, and acute summer diarrhœa.

As examples of true infectious microbes might be mentioned the obligate parasites associated with the exanthematous fevers, as in their case it is evident that they cause a general infection by multiplying excessively throughout the system, after invasion of the body, although still capable of splitting up the living albumens into poisonous products, which may, as in the case of the toxic organisms, be the chief factors in causing the clinical symptoms by which we recognise the disease. These organisms are true parasites, as they grow and multiply chiefly in the animal body; and although all the infectious parasites may not be incapable of independent life apart from the body, their propagation apart from a parasitic existence must be, so far as now known, extremely limited.

With regard, however, to certain organisms associated with what are known as endemic diseases, it may well be doubted whether their saprophytic existence apart from the living body is not for them the most important phase of their life-cycle. The diseases which I would include under the term "endemic" are yellow fever, cholera, enteric fever, dysentery, summer diarrhœa, diphtheria, tetanus,* tubercle, malarial and remittent fevers. Possibly, typhus and relapsing fever should be included also, as they have many analogies with diseases of this class. In discussing this question, which is one of great importance in etiology, we may note the following considerations.

(1.) The *endemic diseases* are distinguished from the more purely epidemic forms by their tendency to attach themselves more or less permanently to certain localities, and some of them have very distinct relations to atmospheric and telluric conditions. Some of them may at certain times, under favouring seasonal or other unknown conditions, take on epidemic extension, as it is called, and become widely prevalent over large or small areas, spreading, however, always from an initial focus, or foci. Whilst personal con-

* The discovery of the bacillus of tetanus in certain soils of different localities places this disease in the endemic class.

tagion plays a very considerable part in the propagation of many of these diseases, especially during the periods of epidemic extension, the cases that arise in the localities where the diseases are endemic are often impossible to trace to a previous case, and, as is well known, the *de novo* origin of many of these complaints has been, and still is, a matter of discussion.

In tetanus, tubercle, malaria, and summer diarrhœa, a theory of origin from personal contagion is not possible, excluding very exceptional cases. These are now known or believed to be zymotic diseases, dependent upon the presence of certain special micro-organisms; but these microbes must be capable of independent life apart from the body, and they, therefore, have a saprophytic existence in certain localities which favour such a mode of life. It may well be in the case of all, as we know to be the case in malarial diseases, that the introduction of the microbe into the animal body is but a fortuitous phase of its ordinary mode of existence, and not necessary in any way for the completion of its proper life-cycle.

Now, with regard to the diseases supposed to be caused by true infectious organisms, those which are mainly propagated by the transmission of infective particles through the air are *epidemic diseases*, which, with the possible exception of influenza, spread by means of personal infection, no other form of contagion being known or sought for than that which arises from a previous case of the disease. The periods intervening between epidemics are probably largely determined by the accumulation of susceptible persons in the population. In large communities, where the aggregation of the population is close, we see measles and whooping-cough tending to become epidemic every two or three years, scarlet fever every five years, small-pox every ten years, and so on, as generations of children spring up who are unprotected by previous attack, or have lost the protective influence of a primary vaccination in the case of small-pox. In large towns these diseases are always, to a certain extent, prevailing in non-epidemic periods, and are to that extent endemic, but the continuity of cases can almost invariably be shown to be solely due to personal contagion, direct or indirect.

Influenza rather stands alone as a disease only witnessed as a pandemic, spreading every forty years or so over a great part of the globe, having doubtful relations with telluric and atmospheric conditions, possibly endemic in certain remote parts of the world, and possibly owing little of its wide and

rapid spread to human intercourse and human infection. It is, therefore, a disease *sui generis*, and need not interfere with the general consideration. Other diseases, as small-pox and cholera, may at intervals become pandemic.

(2.) Another broad distinction between the endemic and epidemic diseases is that the protection conferred by an attack of the former is, with certain exceptions, such as yellow fever, very slight or absolutely non-existent, whereas an attack of one of the eruptive fevers is usually more or less protective for the rest of life. Influenza is, no doubt, an exception, as was recently so strikingly shown in this country; and relapsing fever is also said to confer but a very slight amount of protection.

Whatever be the explanation of acquired immunity, whether it be that the leucocytes of the bodily fluids, having become accustomed to the poisonous products of pathogenic microbes, are able to resist the action of these poisons, and transmit this acquired property to subsequent generations, so that the body becomes, after an attack of infectious disease, full of resistant living particles, active to repel a fresh invasion of microbes; or whether it be that the first attack leaves in the system for an indefinite period a product or substance of such a nature as to prevent the growth of any microbes of the same species subsequently invading the tissues; whatever the view held, it must be that the intimate pathological processes originative of disease in non-protective complaints are very different to those which ensue on an attack of a complaint which is protective against itself. We have already had occasion to attribute the endemic or non-protective diseases to the action of organisms producing toxines, and not multiplying in the system, whilst the protective diseases have been attributed to the action of true infectious organisms. In this direction possibly lies the explanation of the differing degrees of immunity conferred.

(3.) Although, as a rule, an attack of an endemic disease confers little or no immunity from subsequent attack, yet people who reside in districts where yellow fever, cholera, enteric fever, and malarial disorders prevail, undoubtedly enjoy a certain immunity from attack, and are recognised as having become acclimatised. Different races of men exhibit different degrees of freedom from certain endemic diseases, but it is probable that it is not so much a question of race as of residence for long periods in infected districts, for natives of such endemic areas appear to lose their immunity if transported to regions which are not affected, and the immunity can only be reacquired by return to the original spot, or to localities similarly affected.

The experiences in India with enteric fever, amongst the young and freshly-imported troops, will point to this disease as one for which acclimatisation is possible, as well as for yellow fever, malaria, and cholera. With regard to tubercular diseases, it is probable that civilised races also undergo an acclimatisation, which confers upon them a certain amount of immunity, for the tubercle bacillus is known to be very widely spread in the air and dust of towns and villages, yet but few proportionally of the population are attacked. Native races, however, like certain tribes of negroes, South Sea Islanders, and American Indians, who live an outdoor life away from all the influences of civilisation, are found to be wonderfully prone to contract tubercle when transported into cities, and compelled to live under artificial conditions of existence. In the same way we can account for the good health enjoyed by the men working in sewers or employed at sewage works. These men, after a few months of work under such conditions of contaminated air, become habituated to the germ-tainted and often specifically-polluted atmosphere. The bodily forces are then attuned to the combat with even deadly microbes, and they escape from such diseases as enteric fever, diphtheria, and diarrhœa, which are known to be often spread by sewer air and sewage gases. Many of these men, however, after a short trial, are unable to stand the work, and, becoming ill, give it up. Those that remain, therefore, are specially fitted by natural constitution to resist disease invasions, and are, moreover, rendered immune by what we may consider as the continual inoculations with attenuated viri, to which they are exposed when at work.

Now, in view of this faculty of acclimatisation in endemic areas, the question arises, may not the pathogenic micro-organisms of such diseases as diphtheria, tetanus, erysipelas, septicæmia, yellow fever, cholera, enteric fever, acute diarrhœa, tubercle, and malaria, have developed originally from the local saprophytes of the regions in which they occur? And is it not probable that the original less virulent organisms still exist in these endemic regions in the water and earth of the locality, and that the influence which such places exert on their inhabitants depends on the presence of these harmless organisms, which, when introduced within the body, can confer a certain protection, without giving rise to any symptoms of disease?

This is a theory propounded by Professor Hueppe and Dr. Cartwright Wood in an account of investigations made by them into the relations of putrefactive to parasitic bacteria

(*Lancet*, Dec. 7, 1889). In this research it is shown that certain animals may be rendered immune against virulent anthrax by preventive inoculations with ordinary saprophytic bacteria. They found in both water and earth a bacillus which, by its appearance and growth in gelatine, was indistinguishable from the *bacillus anthracis*, but without its physiological or pathogenic properties, since it exerted no action on white mice, which are extraordinarily susceptible to anthrax infection. Of a large number of mice, guinea pigs, and rabbits, which were inoculated with this saprophytic bacillus, and which remained apparently quite unaffected as the result of the inoculation, a considerable number were subsequently found to have been rendered completely immune against virulent anthrax infection; whilst in others which developed anthrax, the disease ran a far longer course than in the control animals not previously inoculated, and the cultures of bacillus obtained from them exhibited marked attenuation.

These researches show that one at least of the saprophytic bacteria has also the latent power of dissociating living albumens, as without this power inoculations of cultures of the saprophyte would be incapable of rendering animals immune against virulent anthrax. If the saprophyte was absolutely inert when introduced into the body, it could not possibly confer any sort of immunity. If this is so, subsequent discovery will probably show that many other saprophytes also possess this faculty. Already it has been proved that one of the common mould fungi—*aspergillus fumigatus*—when inoculated into rabbits, induces a disease characterised by metastatic deposits in various organs; and the *torula*, or yeast fungus, inoculated into monkeys produces a constitutional febrile disease, which is to some extent prophylactic against future infection (Buist). The relation of the septic diseases to the products of putrefaction in organic liquids is also acknowledged to be a very intimate one.

It may well be, then, that certain saprophytes are only distinguished from certain pathogenic microbes by the fact of their possessing in a lower degree only a property which is the essential feature of the latter; and if subsequent discovery should confirm this view, there will be little difficulty in the way of establishing a genetic relation between saprophyte and infective microbe, as we have only to ascribe to the latter, during the countless generations of its evolution, the gradual elaboration of a function with which both are known to be endowed, but in very unequal degrees. As the

microbe acquired greater pathogenic powers, it naturally lost those properties of splitting up the dead albumens in the ordinary fermentative and putrefactive processes, which were the sole active functions of its remote ancestors; and in its highest degree of evolution we now see it as the obligate parasite, entirely dependent for its growth and sustenance upon a parasitic existence in an animal body.

Stated shortly, then, the theory is this: The pathogenic microbes are not specific entities of original creation, but are descended from a common ancestral saprophytic form. They have, owing to gradually altering conditions of environment, developed in successive countless generations the faculty of splitting up the living albumens, and thereby acquired more or less virulent pathogenic properties. The toxic or non-infectious microbe may be but a stage in the passage of the saprophytic to the parasitic and intensely virulent organism, as it is undoubtedly a link connecting the two.

We see a reversion of type in the attenuation of the virus of virulent microbes by appropriate means. The cultures of those microbes which are used as vaccines or protective inoculations are really weakened or degenerated descendants of the virulent microbes—degenerated by exposure of successive generations in culture media to heat, to the action of antiseptics, to drying, etc. (Flügge). It is also remarkable that in some cases the attenuated organisms grow much more vigorously in artificial media, showing the reversion to a saprophytic mode of existence (Wood). They are then, probably, reversions to an original type, yet they must have to a certain extent the faculty still remaining of splitting up the living albumens into toxic substances, inasmuch as they are protective against the virulent forms of the disease—the protection conferred now being believed to be due to the production of the toxine in quantity sufficient to accustom the bodily cells or fluids (globulins) to its action, thus rendering the animal immune, but yet insufficient to give rise to any symptoms of the disease.

Now, what is the bearing of such a theory as this upon curative and preventive medicine? It surely means that if evolution has gone on in the past, it is still in progress in the present. That if new zymotic diseases have appeared within the history of man, still more new ones may be expected to arise with altering social, sanitary, and local conditions, offering different phases of environment to now existing saprophytes. That for the same reason now existing common diseases may in time disappear from off the whole or a

great part of the globe, or may gradually assume characteristics quite distinct from those by which they are now known. That whilst in regard to such diseases as we now consider to be caused by obligate parasites, we should not expect to find any very great differences in manifestation, nor trace them to any but a pre-existing case; in others, which may still be thought of as not having yet attained the highest degree of evolution, we should expect to find various anomalies, not merely in severity, as this can be explained on other grounds, but in type, in infectiveness, in ability to protect from subsequent attacks, and, most of all, in respect of origin from immediately pre-existing cases.

If it be true of the microbes of certain endemic diseases that they are still in an evolutionary stage, and have not yet emerged entirely from a saprophytic existence, we should expect to find not only the anomalies in form, etc., alluded to above, but the infective properties of the microbes cast off from the body retained for great lengths of time by their successors pursuing a saprophytic mode of existence in earth or water.

It is not necessary in cases of disease apparently unconnected with a previous case to assume a *de novo* origin; all that is necessary is to grant that the successors of the original contagious microbes cast off from the animal body have retained the infective powers of their ancestors in a latent but possibly modified condition. To assume a present-day *de novo* origin for any disease appears to be a fallacy, for the element of time—*i.e.*, very long periods of time—is evidently of the utmost importance for the evolution of specialised functions in all classes of plants and animals.

There is still another consideration with regard to these microbes, and that is the possibility of certain partially developed forms existing in nature, which, under certain circumstances, may impart protection without disease arising when they gain an entrance into the animal system, and under other circumstances may produce more or less obscure symptoms of disease incapable of being referred to any known disorder. Much probably depends upon the soil—*i.e.*, the constitution and hereditary proclivities of the animal on which the seed is sown.

Such a theory as this is the flat contradiction of a rigid specificity dogma, but is more in accordance with natural biological science generally, as recognising that amongst microbes, as amongst other plants and animals, the inclusion of every individual amongst certain species divided off from

each other by hard-and-fast distinctions is not always possible. It recognises, too, the immensity of the subject, and does not require that every manifestation of zymotic disease should be capable of being recognised and labelled as something which is already known to human understanding, because it must belong to one of a limited number of recognised species. It also helps to explain much that has puzzled epidemiologists; for instance, the differences in type, in manifestation, and in infectiveness, that characterise certain diseases as seen in temperate and in tropical climates, as seen at the present day, and as recorded in times long past. It is, then, the theory of evolution which is Darwin's immortal conception, and which, in its own department, does not recognise any fixity or unalterability in the constitution or functions of the organic causes of disease, but allows for change in every direction, evolution and reversion, according to altering environment.

The bearing of theory upon practice is often very intimate; but even should the reading of this paper be only regarded as the discussion of a theory, still if it enables us to review broadly the ground on which we stand in our advancing knowledge of microbes and microbial diseases, it may not have been without its uses.

RETROSPECT OF THE SUCCESSIVE EPIDEMICS OF CHOLERA IN EUROPE AND AMERICA, FROM 1830 TO 1890.

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Now that the cholera, which during the last three years has been slowly but steadily advancing westwards from its Indian home, has apparently halted for the present on the very threshold of Europe, it may be interesting and instructive to take a retrospective view of the course of previous waves, and to formulate our conceptions of the factors determining the prevalence and progress of the disease.

I shall not attempt to combat the opinion entertained by men of whose ability I have the highest opinion, but who would themselves frankly admit their unacquaintance with bacteriological work or literature, viz., that cholera is conveyed not by human intercourse but by telluric, meteorological, or other hypothetical agencies or occult influences, unknown, and indeed unknowable.

Nor is it necessary to refute the notion prevalent among the populace of the countries bordering on the Mediterranean that cholera is contagious in the same sense as small-pox or scarlatina, a notion which underlies the practice of quarantine in those regions. I shall assume that is recognised by scientific and competent observers in this country as well as in India, Germany, and America, and by the most thoughtful and independent minds in France and Italy, that its communicability is closely analogous to that of enteric fever; that is, that the virus is a specific microbe present in the evacuations, at any rate in the early stage of the disease, distinguishable by cultivation from some others which in form appear identical with it; that the virulence of the contagion increases for some days after its discharge from the body; that it gains access to other human organisms, solely or almost exclusively by ingestion, and that mere contact with or proximity to infected persons is practically free from danger, though specifically soiled clothes, bedding, etc., may

act as fomites, retaining the poison under certain circumstances in an active state for long periods; that under very unfavourable sanitary conditions of overcrowding amid soiled and infected surroundings, it may enter by the respiratory passages, but that the most frequent modes of infection are "eating with unwashen hands", or drinking water fouled by the excreta of previous cases.

It is thus carried, not by merchandise, but by the persons and clothing of the crowds attending fairs and pilgrimages, caravans, pilgrim, emigrant, and troop ships, if one or more be infected prior to their departure, and it is developed in an epidemic form in any place where the local conditions are such as to admit of the contamination with choleraic evacuations of the soil and water, whether of rivers, reservoirs, or wells.

General insanitary conditions may favour its propagation, but water plays by far the most important part; thus, otherwise insanitary towns, as Rome and Seville, provided with water from sources beyond reach of contamination, have enjoyed an almost complete immunity from cholera, though enteric fever and diphtheria are always present; while, on the other hand, an infected public water-supply may set up a local epidemic where the general sanitary conditions leave little or nothing to be desired, and in this consists one of the dangers incident to the use of rivers as sources of water-supply.

Lastly, while in a few regions of the globe, notably lower Bengal, cholera is never absent, the microbes existing and perpetuating themselves in the soil; there are others, as the remaining provinces of India, where they remain dormant in the soil, ready to break forth into activity at various intervals, depending on seasonal conditions or aggregations of even healthy human beings on previously infected sites; and still others, as in Europe and America generally, where it does not appear capable of maintaining itself for more than a few months, or a year or two at the outside.

Spain, with its subtropical climate, is, I believe, gradually bringing itself under the second of these divisions; and I am convinced that the recent epidemic was a recrudescence or revival of that of four years ago, unconnected with and entirely independent of the "contingent" that is still hovering on the confines of the Red Sea, the Levant, and the Euxine.

The incubation period has been variously estimated at from a few hours to several weeks; the latter assumption is absurd, and the alleged instances are to be explained by

"deferred infection", conveyance of fomites, or by errors of observation. It usually ranges between twenty-four and forty-eight hours, but may be shorter and is rarely longer; indeed, for purposes of quarantine, four days is the utmost reasonable period; six would constitute an extreme limit, allowing for the not infrequent prolongation of the ill-defined and by no means characteristic symptoms of the initial stage, consequent on the feebleness of the infection or the great power of resistance possessed by the individual.

The dependence of cholera on human agencies for its transport is clearly shown by the uniformity with which successive epidemics have followed the regular routes of trade or of pilgrimage, in Persia, Transcaucasia, the Euphrates Valley, Arabia, and Syria, while the rapidity of its march everywhere corresponds to the facilities for travel and intercourse. Where men march on foot in caravans it is slow, where railways exist it advances by leaps and bounds, and where there is much coasting trade, or free communication between neighbouring towns, it diffuses itself in every direction. Dr. De Renzy, writing in 1872, said: "Epidemics of cholera are becoming more and more frequent in the upper provinces of India; and from thence are easily carried over into Persia. The winds are the same, but travel has much increased. Calcutta and Lahore, only twenty years ago, were five months' journey apart, now only five days. Tens of thousands of Hindoo villagers now travel where few did before."

Australia alone, of all the quarters of the globe, has to this day enjoyed exemption from visitations of cholera, its communication with the rest of the world being conducted solely by large and well-appointed vessels, and being mainly with Great Britain, where epidemics have been few and far between. The intercourse with China is under strict supervision; but should an immigration of Indian coolies, comparable to the stream of emigration from Europe to America, ever set in, I am convinced that Australia would soon cease to maintain its unique immunity.

On the conveyance of cholera from Hindostan to Persia, Lieut.-General Sir Alex. Burnes wrote twenty years ago: "The most extensive arrangements have long been made to convey pilgrims and merchandise, and with them cholera, to and from North-West India and Persia. The Lohanee Afghans are a migratory, commercial, and pastoral people, who proceed annually from the borders of Persia down into Hindostan in order to purchase merchandise. At the end of October, as winter approaches, they leave Khorassan in

Persia and descend into India, where they remain until after the great fair at Hurdwar. They commence their return towards the end of April, and all reach Cabul and Kandahar by the middle of June, in time to dispatch their investments to Herat and Bokhara, and then pass on into Khorassan in Persia, where they remain during the summer. They march in three great divisions with 24,000, 19,000, and 7,000 camels respectively."

Due west of Cabul and Herat lies Meschid, the Holy City of Northern Persia. For eight months in each year all the roads to and from Meschid are thronged with pilgrims, some sixty thousand of whom on an average come from India and Afghanistan, and as many from Anatolia, Transcaucasia, and the countries east of the Caspian Sea.

Then there are two other shrines, Mesched Ali and Mesched Hussein in the valley of the Euphrates, which, though somewhat less holy, are in high favour with the Mussulmans of India on account of the greater facilities afforded by the sea passage, the discomforts of which they endure in order to save the time which the tedious pilgrimage through Afghanistan involves. Bad, however, as the accommodation that they can obtain in exchange for a few hard-earned rupees on board the vessels on which they embark at Bombay or Kurrachee may be, it is luxurious in comparison with that of the river-boats to which they are transferred at Bussorah, near the head of the Persian Gulf. Old and young, poor, ill-fed and ill-clothed, already suffering from sea-sickness and exposure, and some probably carrying with them the infection of cholera, dysentery, or enteric fever, are crowded together like sheep in a pen, with no provision for decency or shelter from the burning sun or rain. Many die on the passage, but the dead continue their journey along with the living, the miserable fanatics believing that the salvation of the souls of their friends is equally secured whether they reach the holy place alive or in a state of advanced putrefaction, there to receive a hasty interment.

Lastly, there is the holiest shrine of all, that of Mecca, where the Prophet himself lies buried. To it pilgrims flock from every part of the lands of Islam, from Bengal to Morocco, from Zanzibar to Astrakhan and Khiva, by sea and by land. The Anglo-Indian, Anglo-Egyptian, French and Russian, perhaps even the Turkish Governments, may exert some control over the shipping, but enormous numbers of pilgrims come overland from all parts of Arabia and Syria, the Euphrates Valley and Persia, the well-to-do on camels, the poor on foot. Camped around Mecca, with scanty and

unwholesome food, no shelter from sun or rain, and no precautions to prevent the whole soil from being saturated with the excreta of man and beast, they die by thousands, where only hundreds are officially reported, and the corpses, scarcely covered by the sand, are soon exposed by the wind or rain, and by dogs or birds of prey.

From a comparison of successive epidemics Dr. J. C. Peters has described several distinct and well-marked routes followed by cholera on each occasion. Two, the Central Asiatic and the North Persian routes, start from Cabul; the former proceeding by way of Balkh, Bokhara, Khiva, and Orenburg to Russia; the latter by Herat and Mesched, to Astrabad and Teheran. From Astrabad and from Teheran *viâ* Reshd it is carried across the Caspian to Baku and Astrakhan; while another route from Teheran proceeds overland to Tabreez, where it diverges towards Tiflis to enter Russia, and to Erzroom and Trebizond, to be carried along the shores of the Black Sea in every direction.

The Persian Gulf route, which is followed by almost every epidemic, after touching at various ports on either side of the Gulf, gives off a branch at Bushire, which, passing north-eastwards *viâ* Shiraz and Ispahan to Teheran, follows the route already described as the North Persian; while the main body proceeds up the valley of the Euphrates and Tigris by Bussorah and Baghdad to Diarbekir, and in a north-westerly course to Aleppo, which, though not a seaport, is in close communication with Iskanderoon, Alexandretta, and Latakia.

The Red Sea route, which had not been followed previously to 1865, has ever since attracted the chief attention of the Governments of Europe, with the exception, perhaps, of Russia, on account of the enormous extension of communication with the East consequent on the opening of the Suez Canal, with the increased facilities afforded thereby for the Mussulmans of Northern Africa and European Turkey to resort to Mecca. Alexandria is in constant intercourse with the seaports of Spain, France, Italy, Greece, Turkey, the Danubian States, and Russia, even Marseilles and Odessa being within a few days by steam, while these ports, with Trieste and Constantinople and Smyrna, are again centres of diffusion.

Cholera, which had for two or three centuries been described by Portuguese, Dutch, and French physicians in India, and had from time to time decimated our armies since 1808, first extended itself beyond the limits of Hindostan in 1821, when it ravaged Persia and Arabia, and,

travelling along the shores of the Caspian, reached Astrakhan in 1823. It then subsided, and did not reappear in these lands until three or four years later.

In 1827-8 it again invaded Persia and the trans-Caspian countries, reaching Astrakhan, Orenburg and Perm in the autumn of 1829, and in the course of the following year Tcherkask, Nijni Novgorod, Saratov, Moscow, and most of the great centres of population in the Eastern governments. In 1831 it burst out with fearful intensity, invading the western provinces from Petersburg to Odessa. At this time the Polish revolution broke out, and the massing of troops from infected districts, together with the sufferings inseparable from so desperate and relentless a struggle, developed the disease with appalling virulence. Every effort was made to keep it back from the German frontier, but without avail; and it soon extended over the whole of Germany, Austria, and Western Europe, advancing with such rapidity that its appearances in the most distant places seemed almost simultaneous.

The first cases in England occurred in July and August 1831 on board of some vessels in the Medway, where numerous sail had arrived from Riga during the preceding weeks. In October it broke out at Sunderland and Gateshead among the discharged crews of vessels engaged in the Baltic trade and persons associating with them. It speedily spread to Newcastle and Edinburgh, thence to Glasgow by the canal, and onward to Belfast, Dublin, and Cork, reaching London in February 1832.

Between April 28 and June 3 four vessels, carrying 370 emigrants from Limerick, Cork, and Dublin, arrived at Grosse Isle in the St. Lawrence, after having lost on the voyage no fewer than fifty-nine passengers from cholera. During these three months 30,000 emigrants arrived in Canada, of whom over 7,000 passed through Quebec between the 3rd and 5th of June. These carried the disease in every direction, up the river and lakes as far as Michigan, from Montreal and Ontario to New York, from Erie to Ohio, Kentucky, Pennsylvania, and Tennessee, and from Chicago to the valley of the Missouri and Upper Mississippi. The United States troops, at that time engaged in a desultory warfare with the Indians to the west of these rivers, suffered severely, having contracted the disease from using infected boats.

In 1833, New York was invaded direct from Europe, and became a centre for the diffusion of cholera through the adjacent States, and as far south as Charlestown, while the

disease, which had been raging in Cuba, Vera Cruz, and the Mexican shores, was imported into New Orleans, whence it spread along the southern coast to Alabama and Florida, and up the valley of the Mississippi as it had previously travelled down it. In 1834, cholera again entered the States from Canada, and lingered there till the end of 1835, when it finally died out.

Meanwhile, in India, cholera, though never absent, had been subject to very marked fluctuations. Between 1836 and 1839 it had spread over Afghanistan, Persia, and Arabia, almost reaching the Mediterranean; but retiring again beyond the Indus. In 1840-2 it raged in China, spreading during 1843-5 over Balkh, Bokhara, and Samarkand, and in the following year reinvaded the north-west provinces of India from without, thus reversing its usual course. In 1845 it prevailed in Afghanistan and Central Asia, and in 1846 it burst out among the pilgrims at Mesched, rapidly extending over the whole of Persia; from Tabreez it spread northwards to Baku and the western shores of the Caspian, and from Baghdad southwards to Bushire, whence it was carried by pilgrims to Mecca.

In 1847 it reappeared at Derbent in April, and at Astrakhan in July. From Tabreez it reached Tiflis, spreading thence to Gori and Poti on the Black Sea. From Teheran it was carried by the caravan route to Erzeroum and Trebizond, which it reached in September. A large Russian army was at this time concentrated in the Caucasus, and a great military road was in course of construction between Tiflis and Moscow, through Stavropol, Taganrog, Voronetz, and Toula, all which places were involved in succession. From Astrakhan it followed the course of the Volga, the most important highway of commerce in Russia, attacking every town from Saratov to Kazan, turning south-eastwards to Orenburg and westwards to Nijni Novgorod, where the great fair served to propagate and diffuse it, as does that of Hurdwar in India.

Meanwhile, the cholera was carried from Trebizond to Constantinople and Odessa; and from Constantinople to Smyrna and the seaports of Syria. From Odessa it ascended the Dnieper into Poland, while from both Odessa and Constantinople it was conveyed up the Danube to Roumania, Bulgaria, and Hungary. Here, as in Poland, in 1831, war was raging, though on a still larger scale, and the aggregation of some 800,000 Austrian, Hungarian, and Russian troops fomented the epidemic, which their subsequent dispersion carried throughout Italy and Germany, though it did not reach Paris until March 1849. Early in October 1848

it was carried from Hamburg to London, and a week or two later to Hull and Sunderland, while Edinburgh received it direct from Russia. By the end of 1849 scarcely a town in Great Britain and Ireland had escaped, and the same might be said of every country in Europe, except Denmark, where a most rigid quarantine was maintained. In Paris, 33,000 cases occurred in the three months of April to June; and in London, during August, the deaths numbered 1,200 weekly.

The institution of a house-to-house visitation in London, and the larger towns of England and Scotland, was not only successful in arresting the progress of the plague, but threw a flood of new light on its etiology and mode of propagation. That it was transportable was but too plain to be gainsaid, though the theory of its being contagious, in the usual meaning of the word, was no longer tenable, and was abandoned by the physicians of England and Germany. The belief that the poison was contained in the evacuations, and that it was propagated by the contamination therewith of water, clothing, bedding, or food, advocated by Sir J. Simpson and Sir R. Christison of Edinburgh, Dr. Kirk of Greenock, Dr. Parkes of London, Prof. Von Gietl of Munich, and W. Wagner of Berlin, began to gain adherents on all sides; while the connection between it and the premonitory diarrhœa was recognised by many who, as yet, hesitated at accepting this explanation of its causation and spread.

The relation between cholera and insanitary surroundings was ably shown by Dr. Southwood Smith and Sir Edwin Chadwick in the Report of the Royal Commissioners, which revealed a state of things existing in the metropolis which in the present day would appear incredible. The whole of Lambeth and Bermondsey was intersected by "open ditches of the most horrible description". St. Olave's and St. Thomas's were "a disgrace to the civilised world". In Whitechapel "there was neither sewerage, drainage, cleansing, nor a good supply of water". "The characteristics of the streets of Hackney were overflowing cesspools and privies, cowyards and piggeries, with a loathsome ditch." The state of Battersea, Plumstead, and other low-lying districts was dreadful; and even in parts of Kensington, Shepherd's Bush, and Hampstead, things were little better.

The circumstances attending the introduction of cholera into the States in this year possess, both from an etiological and prophylactic standpoint, an interest which cannot be overestimated. Two ships, the *Swanton* and the *New York*, left Havre on Oct. 31 and Nov. 9 respectively, with German emigrants, the former bound for New Orleans and the latter

for New York. Havre was at the time free from the disease, and the vessels consequently received clean bills of health; but many of the passengers had come from infected districts in Germany, Hungary, and Poland. Cholera broke out on board the *New York* on November 25th, when she had been sixteen days at sea, and on the *Swanton* on Nov. 26th, or twenty-seven days after her leaving Europe. One was in latitude 42°, the other in 25° 47′, or a thousand miles apart. It is absurd to suggest that the disease had been contracted in Europe, for even if one should admit the possibility of an incubation period of over sixteen days, no one has yet imagined that it could be prolonged beyond a month; while, considering that there was no cholera on the American continent, and that a strong south-east wind was blowing for some time before the outbreak, it is hard to speak with courtesy of those (and there are some) who pretend that the infection was conveyed aerially! This wind was extraordinarily hot, but preceded, in the case of the *New York*, by a cold, chilly wind, during which there was, in the words of the captain, "a general overhauling of baggage for warm clothing." One passenger was known to have in his trunk clothing that had belonged to a man who had died of cholera in Germany. The obvious and only rational explanation is that the infection was contained, and retained its vitality, in the closely-packed and unventilated baggage, the opening of which was brought about by the sudden and extreme changes of temperature. The *New York* arrived at the city of the same name on Dec. 1st, and the *Swanton* at New Orleans on Dec. 11th; each vessel having in the interval lost several passengers from cholera. The disease in each case was carried by the new-comers in different directions; at New York it subsided during the severe cold of January, but at New Orleans the deaths rose steadily from 400 in December, and 600 in January, to 2,500 in June 1849. During December other vessels arrived from Germany at New Orleans and other ports with cholera on board, thus keeping up a succession of imports of infected persons and things, while the dispersion of the immigrants, the flight of terror-stricken citizens, and the ordinary coasting and internal trade carried the pestilence to places as remote as Cuba, Sacramento, and San Francisco. It died out, however, before the commencement of the year 1850.

The widespread epidemic of 1854, which extended to Great Britain and the United States, and is commonly reckoned by the medical historians of these countries as a separate visitation, was, as a matter of fact, not the consequence of any

fresh invasion from the East, but merely a resuscitation or exacerbation of the previous epidemic, which had never entirely died out on the continent of Europe, summer or winter, from 1847 to 1859, reinforced by occasional importations from Asia *viâ* both Russia and Egypt. In 1850 it had almost died out, though it lingered along the Polish frontiers of Germany and Austria, especially in Bohemia, but it broke out in 1851 in Berlin and Vienna, Trieste and Malta, Pesth, Marseilles, and Northern Italy, though the rest of Germany, Holland, and France remained free. It persisted in Russia and Poland, and had raged at Mecca and Cairo. In 1852 it was confined to Russia and Eastern Prussia, but in 1853 it spread westwards over the entire continent, scarcely abating even in the depth of winter. England was invaded late in the autumn by arrivals from Hamburg and the Baltic ports; London, Liverpool, Manchester, and Edinburgh suffering most severely. Twenty-eight infected vessels left England, Holland, France, and Germany for America, with, together, 1,141 deaths on board during their passage. It continued to rage in Persia, and in 1854-5 it decimated the population of every town in Europe and the allied armies in the Crimea; while Denmark, Switzerland, and Greece, which had hitherto, thanks to strict quarantine regulations, been exempt, were attacked for the first time—an immunity totally inconsistent with any hypothesis of the transportation of the disease by winds or "epidemic waves". In the case of Greece its introduction was evidently by means of the troopships of the allies. It is nearly certain that in America and on the continent of Europe cholera continued to linger, especially in New Orleans and the Mississippi Valley, from 1849 to this time, while infected arrivals from Europe set up small local epidemics at different places in Canada and the States in every intervening year. But, as I have already mentioned, the great importation which may be looked on as the starting-point of the general epidemic took place in November 1853. The first persons attacked were the emigrants landed from ships on which cases had already occurred; but though they disembarked at New York, they did not manifest the disease until their arrival a few days later at Detroit and Chicago, when, as Dr. Alonzo Clark observes, they first unpacked their infected luggage. During the early months of 1854, similarly cholera-smitten vessels continued to arrive, and the railways and river-boats conveyed, first, infected immigrants, and afterwards infected citizens, to the remotest cities and States, while from New Orleans the disease was exported to the West Indies and Mexico, Jamaica and Cuba suffering most severely.

For ten years, however, both Europe and America enjoyed repose, and the next visitation is remarkable as marking a new epoch in the history of cholera, viz., its introduction by way of the Red Sea, wholly or in great part. I am far from asserting that such importations had not occurred from time to time, for it is in the highest degree improbable that with the awful outbursts of cholera at Mecca and with the annual fairs or festivals at Tantah, in Egypt, where from half-a-million to a million persons of both sexes mix in horrible orgies at the tomb of Said el Bedoui, and whence the cholera had several times spread over the whole of northern Africa, it was not carried to Europe; but prior to the inauguration of the liberal policy of the late Ibrahim Pasha, the opening of the Egyptian railways, and above all of the Suez Canal, the intercourse between Egypt and the countries of Europe was utterly insignificant compared with what it is now, while the the canal has brought the privilege of a visit to Mecca within reach of every Moslem in the countries bordering on the Mediterranean and Black Seas. If we except a few isolated cases at Malta, cholera had been unknown in Europe since 1855, at any rate west of Russia, until on June 11, 1865, it was brought to Marseilles by some pilgrims returning to Algiers. In this town the deaths rose from sixteen in June to 768 in December. From Marseilles it was carried to Paris and Havre, to Naples, Valencia, and Barcelona, while other vessels from Alexandria conveyed it to Ancona, Smyrna, Jaffa, Beyrout, and to Constantinople, which, constituting a second centre, communicated it to Varna, Galatz, Trebizond, and Odessa, from which last port it penetrated into Russia. From Galatz it passed up the Danube, invading Roumania, Bulgaria, and Hungary, and by the end of the year the whole of Europe was again involved. In England, however, beyond a few cases at Southampton, it did not appear until May 1866, when it broke out among some German emigrants *en route* to America; and in June in London, where the earliest cases were those of recent arrivals from Hamburg and the Baltic. No sooner, however, had it obtained a footing than the weekly mortality rose from sixteen in the last week of June to 1,053 in the first of August, when, thanks to an energetic house-to-house visitation and sanitary measures, it as rapidly declined. The total deaths reported in London from June to December were 5,548, of which half occurred in the second month of its progress. Elsewhere the mortality was comparatively light, and the smaller towns and rural

districts escaped entirely. The connection between cholera and the water-supply, first recognised in 1854, received in this epidemic conclusive demonstration, and the British Isles have ever since enjoyed absolute immunity.

In November 1865, cholera was introduced into Guadalope from Marseilles, and the *Atalanta* arrived in New York from Havre with cholera on board, but the consequences were averted by quarantining the ship.

On March 28th, 1866, the *England* left Liverpool for Halifax with eighteen saloon passengers, 1,185 Irish and German emigrants, and a crew of 120. Four days later, one death from cholera occurred, but the weather being rough, tho hatches over the steerage were battened down. The consequence of this cruel procedure was that, on her arrival six days after, forty-six of the emigrants had already died, and though the sick were then transferred to a hospital ship and the healthy placed in tents, the subsequent deaths raised the total to 267. None of the saloon passengers or crew were attacked, although they remained on board the *England*. The *Virginia* and the *Peruvian*, also from Liverpool, lost respectively 116 and 115 passengers from cholera. The whole number of vessels arriving in New York with cholera on board between April and November 1866, was eighteen, carrying 8,491 persons, of whom 872 died of cholera before or soon after landing. No accurate information was obtainable from other ports, but it is certain that the epidemic was propagated from the various points of arrival and from those in the interior at which emigrants assembled before proceeding to their ultimate destinations; while the unsettled state of the country at the close of a long war, with the massing and movements of large bodies of troops, presented the most favourable conditions for the dissemination of the disease.

While the last epidemic was expiring in Europe another invasion was looming from the Far East. From 1867 to 1869 cholera raged in North-West India, a terrific explosion having followed the great fair at Hurdwar in the first of these years, and extended as far as Teheran. In 1868 it was revived by the pilgrimage to Great Mesched, and before the close of the year had reached Astrabad and Reshd on the Caspian. In 1869, Persian traders brought it into Russia as far as Nijni Novgorod, and in 1870 it was introduced simultaneously by the North Persian route *viâ* Tiflis and Poti to Taganrog, Kertch, Rostoff, and Odessa. A fresh outbreak occurred at Nijni Novgorod during the fair in September 1870, where 200,000 traders were assembled from Persia, Central Asia, and all parts of Russia, the latter

carrying it to their homes throughout the empire. From Odessa it spread also to Kief, Moscow, and St. Petersburg, Warsaw, Dantzig and Königsberg, and westwards through Roumania and Hungary to Austria and Bohemia, and from Hungary to Northern Italy, while steamships from Genoa carried it to Brazil and Rio. Constantinople escaped owing to rigid quarantine until 1871, but it was then carried from that city to Alexandria. Its westward progress had been checked by the suspension of intercourse between Germany and France during the war, but at its close, France, Spain, and Portugal, in fact, every country in Europe, was involved. Cholera ships from the Baltic and North Sea ports, from Havre, the Mediterranean, and Constantinople, arrived in London, Hull, Cardiff, and other English ports, but the judicious measures adopted by the British Government were in every instance successful in averting the danger.

In like manner the arrival of cholera-smitten vessels at Halifax, New York, New Orleans, etc., during 1872 was followed by no results; but in 1873 these precautions broke down, and cholera extended over the whole length and breadth of the land. It would appear that in the majority of cases it was not by means of persons actually suffering from the disease, or by ships on which cases fatal or otherwise had occurred during the voyage, but by means of infected clothing, that the importations took place—a danger which it was almost impossible to avert. Thus, to quote from the report of the supervising surgeon of the Mercantile Marine, Dr. Woodworth: "In 1873 three distinct outbreaks of the disease occurred at widely remote points in the United States, from poison packed and transported in the effects of emigrants from Holland, Sweden, and Russia. These people, and the vessels in which they were carried, had been perfectly healthy, and the people remained so until their goods were unpacked at Carthage, Ohio; at Crow River, Minn.; and at Yankton, Dak., respectively. Within twenty-four hours after the poison particles were liberated the first cases of the disease appeared, and the unfortunates were almost literally swept off the face of the earth." These instances, which might be multiplied, sufficiently demonstrate the almost insuperable difficulties incident to the circumstances of a country like America, and which do not present themselves in England, where emigrants from infected continental States merely call *en route* without unpacking their baggage. But in one way or another foci of infection were set up in countless centres, whence the disease was propagated in all directions, though the efforts at their suppression

by stamping out each local outbreak were so far successful that the epidemic did not maintain its hold on the population, or even linger beyond the end of the year in which it began.

In Europe it smouldered in various places, breaking out from time to time until the latter part of 1874, when it seemed to have completely exhausted itself. In 1881 cholera was unusually severe at Mecca, and again in 1883, when it assumed an epidemic form in Egypt, without, however, extending further.

It was in June 1884 that the first cases in Europe appeared at Marseilles, or more probably at Toulon, the infection having been introduced by troopships from Saigon, where it had long raged. From Marseilles it was carried to most parts of France, though the mortality was heaviest in the south. In three months over 5,000 deaths had been reported, half at least from Marseilles itself. Thence it spread to Italy and Spain, and in a less degree to Algeria. The first cases in Italy were at Spezzia, but soon almost every town in the country was attacked, and the people were panic-stricken. At Naples the mortality was appalling, as also at Palermo. Rome alone escaped, having the advantage of an unimpeachable water-supply from the ancient aqueducts. The deaths throughout Italy up to November, when the epidemic was practically extinct, were estimated at between 11,000 and 12,000.

In Spain it appeared first, and was, as usual, most severe in the province of Valencia and Murcia. In Madrid the deaths were 1,366, or not more than occurred in a couple of days in 1865, before the introduction of a pure water-supply. Seville, with water laid on by a British company, would have escaped as completely as Rome, had not the people in the suburb of Triana used the river water for some time; and in Toledo the disease subsided so soon as the Governor compelled the inhabitants to resort to springs outside of the town, placing armed sentries along the river banks. On the other hand, at Granada, one in eight of the population died, and in Saragossa the mortality was not much less.

In Germany, where in all previous epidemics the mortality had been very high, only a few isolated cases were, as in England, observed among persons arriving from France or Italy, owing evidently to the great improvement in the sanitary conditions of the towns, and especially to the general introduction of public supplies of pure water.

In 1885, cholera lingered in its old haunts in France and Italy, but the only serious outbreak was in Spain, where it reappeared at Jativa, in the province of Valencia, and spread

to other towns in the eastern provinces; and the same may be said of the following year, though there may be some doubt as to many of the alleged cases elsewhere.

Europe had remained wholly free from cholera until early in the summer of the year 1890. While all eyes were directed to the slow but steady onward progress of the disease in Persia and the valley of the Euphrates, it suddenly and unexpectedly appeared in the province of Valencia. Mecca had not as yet been reached, Egypt was free from suspicion; in short, the circumstances of the outbreak, which originated in a small inland town, precluded all notion of its having been imported from abroad, though its true character admitted of no dispute. It was ascribed—and I am disposed to believe rightly, or at least very probably—to the opening of some excavations in a site saturated with excreta since the last epidemic. Be this as it may, it is impossible to resist the conclusion of its indigenous origin. Such resuscitation of old foci is familiar enough in Indian experience, and when one reflects on the subtropical character of the climate of southern and south eastern Spain, and the pollution of the soil in and around the towns, there seems no reason to question the probability of the "naturalisation", so to say, of cholera there, as in N. W. India, Persia, or Arabia, where, though not strictly endemic, it appears to be capable of retaining its vitality in a dormant or latent condition for several years, waiting some favourable circumstances for its revival. At one time it threatened to become general, but, by the end of the year it had died out, and for the present, at any rate, the danger may be considered as past.

Meanwhile we must look eastwards, where the history of the earlier invasions seems about to be repeated. Advancing by the old routes through Northern Persia and the Euphrates Valley, it has long since reached Tabreez, Reshd, and Derbent in the north. It has hung for two years around Baghdad, and has travelled up the river as far as Diarbekir, if not to Wan, while it has more recently followed the caravan road to Aleppo. At Mecca the mortality was almost unprecedented, probably tenfold as great as reported; though Egypt almost miraculously escaped.

Tabreez is on the highroad to Tiflis and Trebizond; Reshd and Derbent are in constant communication with Astrakhan, and Aleppo with Alexandretta, Latakia, and Beyrout, in which towns, and in the surrounding districts, cholera has been present, more or less, since the autumn, the weekly mortality, even in January of the present year, having been such as in Europe would have been considered alarming;

so that we may expect with the return of another summer that the history of the former epidemics of '48 and '54 will repeat itself.

Though quarantine may have been at times successful in special circumstances, it is impracticable in Europe, whether on land or in inland seas; but the happy experience of Great Britain since 1866, of Germany since 1874, and of single towns like Rome, Seville, Lyons, and others, has shown that in pure water-supplies the only safety lies, though general sanitation enhances the probability of escape.

THE RELATION OF MODIFICATION OF FUNCTION OF MICRO-ORGANISMS TO THE VIRULENCE AND SPREAD OF SPECIFIC INFECTIVE DISEASES.

By G. SIMS WOODHEAD, M.D.

(*Read: April 15th*, 1891.)

Dr. Louis Parkes, in a paper read before the Society on 18th February, considered the relations of saprophytic to parasitic organisms, and referred to Hueppe and Wood's observations that some of the saprophytes can be distinguished from certain pathogenic microbes only by the fact that they possess in a lower degree, merely, a property which is the essential and prominent feature of the latter—the power of disassociating living albumens, and of producing, though in a very minor degree, certain specific products. Dr. Parkes accepts the theory that pathogenic microbes are not specific entities of original creation, but are descended from the common ancestral saprophyte. By a process of long and gradual acclimatisation or adaptation, certain species have become so altered—either temporarily or permanently—that they are able to exist as parasites, but that the tendency to revert to the saprophytic or non-pathogenic condition is more marked than their tendency to become transformed into the parasitic and pathogenic condition. These suppositions have for some time been advanced as affording explanation of many of the facts bearing on the modifications of the virulence of the specific infective diseases, and I have thought it well to collect and place before you to night a few of the more prominent examples of modification of function of an organism. I say modification of function, because it will at once be accepted that the delicate metabolic changes in an organism are almost invariably brought into prominence long before the coarser morphological modifications can be observed.

In the first place we must look upon a micro-organism, whatever be its nature, as a mass of protoplasm which possesses certain general functions, through the exercise of

which it is enabled to obtain nutriment from animal or vegetable matter. These general functions, however, may be, and usually are, specialised in certain directions according as this mass of protoplasm has to derive its oxygen, carbon, and hydrogen, from sugar and bodies of that class, and its nitrogen or ammonia from proteid substances and the like, or whether it obtains its oxygen, and perhaps even its nitrogen, from the atmosphere. The aerobic growth of an organism, for example, depends on something more than a fixed function or power in any specific organism: it depends on the nature of the food presented to it and on the readiness with which certain foods can be assimilated and certain elements separated. An organism that is for long placed under one set of conditions and receives one kind of food may become so accustomed to this kind of food and to these conditions, that when they are suddenly cut off it may be no longer able to exist, although if the transition be made slowly so as to allow of the protoplasm developing those latent powers that it has of deriving its substance from the new foods and under the new conditions, it may become gradually acclimatised and still continue to flourish luxuriantly. As might be expected, the metabolic products vary with the modifications of food supply and modifications of function, and it is with these that we shall have to deal in attempting to prove that these modifications of function and of environment play an exceedingly important part in determining the virulence of certain organisms that produce specific infective diseases. The first organism on which this modification of virulence was observed was the Bacillus anthracis, the first observations on this being communicated by Greenfield to the Royal Society, London, about the middle of the year 1880. He found that by cultivating the specific bacillus through several successive generations in the aqueous humour of the eye of an ox, he was able to obtain a virus so modified that when injected into animals susceptible to the action of the ordinary anthrax bacillus, these animals did not die; in animals injected with similar cultures carried on to the twelfth generation, no anthrax symptoms at all were developed, the organisms appearing to have lost their power, not only of growing in the blood, but even of producing sufficient of their specific poisonous substances to give rise to any symptoms of disease. Just a month later this modified anthrax virus was utilised by Toussaint to protect an animal against the action of the virulent organism. This investigator concluded that the same principles that applied in the case of Jenner's vaccine virus might also hold good in connection

with the modified anthrax poison. This modification of the virulence of certain organisms has been made use of in all the vaccination experiments that have yet been made, the experimenters going on the principle, so long recognised in connection with the specific fevers, that one attack, even a mild one, usually protects an individual against a future attack of the same disease. With this, however, we are not specially interested, we have here to determine what changes take place in disease-producing organisms when they are grown under conditions different from those in which we have usually to deal with them. This special modification of pathogenic power may take place in two directions: either the organism loses its power of developing in living tissues, or it loses its power of developing a virulent specific poison. For example, the cholera organism cultivated outside the body, as in the case of the anthrax bacillus, usually loses much of pathogenic power. By appropriate treatment, however, such as cultivating through a number of generations at the body temperature in specially prepared broth, its pathogenic—or, rather, its toxic—activity may be restored, such modification in its activity being due to the changes that are observed in the end products, or toxines, that it is now able to produce.

If, however, the same organism be grown anaerobically, although it is able to produce much larger quantities of the specific cholera poison, it is not nearly so resistant to the fluids of the stomach as if it has been allowed to grow aerobically. Cartwright, Wood, and I, in a series of experiments with the cholera group of bacilli, found that under certain conditions there was the formation of sulphuretted hydrogen in very considerable quantities, whilst under other conditions the production of sulphuretted hydrogen was absent. We found, for instance, that when grown on bouillon, Finkler and Muller's bacillus formed sulphuretted hydrogen, whether air was present or not, whilst the cholera organism and Deneke's bacillus produced it only when air was excluded. On yoke of egg and coagulated blood serum all the four organisms of the group produced sulphuretted hydrogen, both aerobically and anaerobically, whilst on white of egg, the cholera bacillus alone was able to set free sulphuretted hydrogen, and this, too, only when air was excluded. If, however, the egg albumen be modified by coagulating it, we have a very striking result in the presence of air—none of these four organisms produce sulphuretted hydrogen—whilst when air is excluded sulphuretted hydrogen is formed sometimes in very considerable quantities. If of this group we

take only the cholera organism we shall find that the production of cholera poison goes on parallel with the production of sulphuretted hydrogen, for Scholl has pointed out that by inoculating an egg by Hueppe's method, *i.e.*, by inoculating an egg and then sealing it so as to cut off a very large proportion of air, the cholera organism acting on the crude albumen is able to produce a very large quantity of the specific cholera poison; whilst, if this same organism be now again cultivated on gelatine, less and less of the specific poison is formed in successive generations.

This briefly indicates the kind of modification of conditions to which I refer when I speak of modification of function of micro-organisms. It is, of course, usually held that the Comma bacillus is entirely an aerobic organism, and there can be little doubt that its vegetative activity is greatest and most vigorous when it is grown in the presence of the oxygen of the air. It is, under these conditions, certainly more resistant to the action of germicidal agents. It must be noted that it does not cease to multiply even when its supply of free oxygen is entirely cut off; the bacteria are, however, much more sensitive to external influences, and are, undoubtedly, much more readily destroyed by acids and other germicidal agents. Wood has pointed out that where free oxygen is cut off (as Pasteur had observed in the case of other ferments) the cholera bacilli produce a relatively much larger quantity of the specific poison or toxine than when oxygen is present, and he shows that when they are grown on albuminous substances with complete exclusion, not only of oxygen, but of all substances from which oxygen can be easily derived, the cholera poison is produced more energetically and more rapidly than under ordinary conditions of aerobic cultivation, and he explains this—an explanation that is confirmed by Scholl's researches—that it is necessary that much larger quantities of albumen must be split up in order to meet the requirements of the organism, and that consequently the resulting metabolic products are present in increased quantities.

Let us see what bearing such researches as these have on the various theories of cholera, and how they may serve to explain some of the apparently anomalous and conflicting results obtained by different workers. Taking cholera itself, we have Pettenkofer maintaining that the increase of cholera is due to the increase of the "drying zone" near the surface of the soil, *i.e.*, the lowering of the level of the "ground water", and that the rise and fall in the level of this ground water is the principal factor in the production of conditions necessary

for the outbreak of epidemics of various kinds. Hence he has naturally taken up a very strong position against the possibility of the spread of cholera directly from patient to patient. There can be little doubt that many of Pettenkofer's observations are entirely in accordance with this view; but it is equally certain that all his interpretations of the facts he has collected are not to be explained on his theory, even though the facts themselves be perfectly accurate. The instances are almost innumerable in which there has been a most remarkable immunity against the direct infection of those in immediate attendance on cholera patients. Even where the dead bodies have been buried at once, the disease has frequently broken out afterwards, not in those who have been engaged in the actual interment of the dead cholera patient, but in those to whom has fallen the duty, a day or two later, of washing the soiled linen used by the patient during life.

How can these facts be explained? In the first case the attendant was exposed to the action of the bacillus as it came from the patient, when, although it was an anaerobic organism, and was in a position to form large quantities of the specific cholera poison, it was nevertheless much less resistant to the action of germicidal agents or to mere drying; it was virulent in the body, and, so long as the conditions were favourable for the production of large quantities of the cholera poison, it could by its presence give rise to very severe symptoms:—Here, in assuming the more specific poison-forming functions, its general powers of resistance were considerably impaired. On the other hand, the bacillus, finding its way on to damp sheets or on to damp soil, remains alive for some time, successive generations are born, which in the presence of air gradually become accustomed to their new surroundings, and acquire greater and greater general powers of resistance; consequently the organism eventually becomes much more dangerous. It may not now be able to form the same amount of poison, but the attendant who a day or two after the death of the patient has to wash the soiled linen may be attacked by the bacillus after it has had time and opportunity to assume its aerobic and more resistant habit, when it is in fact better qualified to live outside the body, to resist the action not only of ordinary germicidal agents, but even of the acid gastric juice, of the stomach of a fresh host. Once it has gained a foothold in the body of its new host, modification in the opposite direction takes place, the organism becomes anaerobic and toxin-producing, but far less resistant.

But this question of modification of function may be carried still further: it may be argued that when Pettenkofer's drying zone becomes limited, the cholera bacillus dies out more readily, and it appears that the anaerobic cholera bacillus passing directly from the faeces into very damp soil—so damp, that it can hold insufficient oxygen to satisfy its saprophytic requirements, remains an anaerobic organism, a form in which it is much more readily and rapidly killed. Where, however, the depth of the drying zone increases, the soil contains much more air (oxygen), in the presence of which the organisms are better able to multiply, and, taking the field against other putrefactive organisms, they gradually become more and more hardy, acquire the resistant aerobic and saprophytic habit, and become more dangerous to the inhabitants of the locality in which all this occurs.

Of course the term drying zone used by Pettenkofer is entirely a relative one, and a drying zone may still contain (as it usually does) sufficient moisture for the wants of the organism. That the cholera organism requires some little time to enable it to pass from the aerobic to the anaerobic condition was evident from Koch's early experiments, in which he made plate cultivations of cholera bacillus, and then, before the gelatine was perfectly set, covered about one-third of the surface with exceedingly fine glass (cover glass thickness) or split mica; he then found that colonies grew as usual, and became visible to the naked eye in the uncovered portion of the gelatine, and for a very short distance under the covering plate (2 mm.). He observed, however, that when the air (oxygen) was cut off, the colonies did not increase in size sufficiently to allow of their being distinguished without the aid of a lens; the power of growth, to the size of a colony visible to the naked eye, being attributed by Koch to the presence of the small quantity of available oxygen that remained in the nutrient gelatine, growth ceasing as soon as this oxygen was exhausted, the colony remaining of small size.

As we have already stated, it may be assumed that the organisms do not require to obtain free oxygen from the air in order that they may continue their growth, for if, as has been pointed out, the cholera organism is grown on a suitable medium, such as unchanged normal albumen, it can not only exist, but can produce unusually large quantities of its specific poison. It is able, in fact, by the disassociation of such a medium, to obtain all that it requires for its growth and development, as a result of which we have the specific or metabolic excretory products that give rise to its character-

istic reaction within the body. Numerous other examples and proofs of this in connection with the cholera organism might be adduced, but the points already mentioned will be sufficient to indicate the lines on which it is necessary to carry on our researches in connection with the relation of the modification of function of organisms to the virulence of epidemic diseases.

Let us take another example of an organism in which modification of various factors in its existence may affect its power of setting up disease. The tetanus bacillus for long eluded the search of the bacteriologist. From the nature of the disease it was very early assumed by Sir James Simpson that tetanus was something akin to hospital fever. It was gradually demonstrated that a certain organism was very frequently met with in wounds, but only in wounds, that seemed to have some relation to the tetanic condition. Most of the early experiments with the organism that was supposed to bring about this condition were extremely unsatisfactory, and considerable doubt was cast on it as a producer of tetanus from the fact that in many cases pus that was known to contain the specific bacillus, and even pure cultivations of the organism, failed to set up the characteristic symptoms when inoculated in the ordinary manner, although the typical bacillus obtained from garden soil, especially when introduced along with the soil, is fatal in a large proportion of cases. Some observers have insisted that the tetanus bacillus, like many others of the septicaemia group, is virulent only so long as it is growing under anaerobic conditions; others have indicated that anaerobiosis is only one of the essential conditions; that tetanus is never produced unless spores have developed in the rods, the formed rods never becoming acclimatised to the absence of oxygen, although the spores, under certain conditions, may develop even in the presence of very small quantities of oxygen. Many of the earlier experiments of inoculating the pus from tetanic patients gave entirely negative results, and it was supposed that such failure was due, in many cases at least, to the fact that the pus, with its contained organisms, had been exposed to the air for some time, and that consequently the bacilli had been compelled to grow under conditions unfavourable to the retention of their specific virulence. In these cases, however, it was almost invariably found that the spores were imperfectly developed, so that we had something more than a mere modification of function: there were distinct morphological modifications. In the case of the tetanus bacillus the modifications as regards the presence of oxygen and of various kinds of

food, and their relation to specific products, have recently had additional light shed upon them by Sanchez, Toledo, and Drs. Vaillard and Vincent, the latter of whom have arrived at the conclusion that it is usually the introduction of the tetanus poison (which they compare to snake poison), and not the inoculation of the organism itself, that gives rise to the tetanic symptoms in animals that are affected experimentally, but that in artificial cultures the organism may grow for a considerable length of time before it acquires this power of setting up the specific disease. This was readily accounted for when it was found that no poison is developed for a considerable time after the organism has begun to grow; the formation of this poison apparently going hand in hand with the formation of a peptonising enzyme which brings about more or less perfect liquefaction of the gelatine.

Even spores, when injected alone, have not the power of setting up tetanic symptoms, but, when they are injected along with other organisms, such as the lactic acid bacillus, or even the products of this bacillus—lactic acid—with the bacillus prodigiosus or into a bruised wound, or where they are injected along with a quantity of their own poison, tetanus is invariably set up. Tetanus organisms, then, forming their own poison slowly, are, in ordinary healthy tissues very rapidly destroyed by the tissue cells long before they have time to form sufficient poison to produce the nervous symptoms of tetanus; whilst, where other materials are injected along with them, the tissue cells are so engaged in removing the other foreign matter, or are so paralysed by the action of the lactid acid, or the minute portions of tetanus poison introduced along with them, that they are not able to contend on equal terms with the tetanus bacillus, which under the favourable conditions by which it finds itself surrounded, grow rapidly, gives rise to the formation of the special poison, and kills the patient. The general bearing of one fact of this nature on the work of the hygienist and bacteriologist is enormous, and the consequences of its acceptance are so wide-reaching that most observers have, justifiably, been very chary of accepting these facts or theories until the evidence has become almost overwhelming.

It has sometimes been objected that these modifications of function exist only in the imagination of the observer, and it has been very pertinently asked what proof exists (beyond what we assume takes place) of the differences in the action of organisms morphologically identical, but physiologically different, on animals or upon the human subject,

and it is difficult to give a complete answer. When we find, however, that actual differences may be observed in the production of colour, for example, we feel that we have much more reliable and convincing arguments at our disposal.

When Dr. Ruffer was working with the Pyocyaneus bacillus, I handed him some nutrient gelatine containing glycerine, without mentioning to him the fact that this was the case; he came to me, and said that he could not obtain the pyocyanin coloration, although he had luxurious growths of the organism. I then remembered that I had given him the glycerine jelly, and we concluded that the absence of the colour must be due to the presence of the glycerine. This was afterwards proved to be the case, for when the original culture was grown on ordinary peptone meat jelly the typical colour was again produced. I was, therefore, very much interested when I found that M. Gessard had worked out the relation of alimentation of the bacillus pyocyaneus to its colour-forming function. Briefly stated, his results are as follows:—The bacillus pyocyaneus grown in bouillon gives rise to the formation of pyocyanin and a green fluorescence; when grown on egg albumen, the functions of the protoplasm are so interfered with that the pyocyanin is no longer formed —we have only the green fluorescence—in peptone gelatine media the fluorescence is lost, but pyocyanin is formed in considerable quantities; whilst, on the addition of glucose to this gelatine medium, the growth of the pyocyanin is also interfered with. He found that by continuing the growth for a series of generations on these various media he was able to eliminate or perpetuate one or other of the colour-forming functions. By the use of antiseptics, or of heat, and by passing the organism through various animals, he was enabled to obtain more or less permanent modifications of these functions—so permanent, indeed, that he looked upon the organisms in which these modifications were obtained as actually forming "race" groups or types.

As these changes can be followed with the eye, it may be well to note the conclusions at which M. Gessard arrives. The pyocyanic bacillus, he says, has at least two chromogenic functions, one of which, the pyocyanic, is perfectly characteristic of this organism; whilst the second, the fluorescent, is common to this and to several other microbes: bacillus fluorescens putidus, the fluorescing bacillus, etc. Both these colours may be formed when the organism is grown in broth; the addition of peptone to the broth renders the development of the pyocyanic function much more marked, whilst it interferes to a certain extent with the fluorescent

function. The addition of albumen assists the fluorescent, but interferes with the pyocyanic, function; whilst, as we have seen, the addition of glycerine interferes with both. If we can apply what we see here to what we observe in tetanus, we at once see that in tetanus, too, we have a specific function, or the formation of the tetanus poison; whilst we have at the same time closely associated with it—in fact, as yet we have been unable to separate it, except accidentally—the enzyme function, a function, however, that this organism has in commonwith very many other organisms, both pathogenic and putrefactive.

Under certain conditions—the addition of glycerine, for example, to the nutrient gelatine medium—many of the enzyme forming organisms no longer produce liquefaction of the gelatine, and we may, therefore, assume that they are no longer exerting their enzyme function; and it is quite possible that, along with the deletion of this more independent general activity, the specific function may also be interfered with. These questions are usually considered from the point of view of the possibility of obtaining an organism which shall have its virulence so modified that it may be used as a protective vaccinal agent; but, as epidemiologists, we are bound to consider this matter on a much wider basis.

The researches of Loeffler, Roux and Yersin, and Klein, taken in conjunction with those of the earlier workers at diphtheria, explain how, in the case of this disease, there is during the course of an epidemic an alteration in the virulence of the disease at different stages, as well as a coexistence of cases of every degree of severity, that can only be accounted for on the supposition that we have a corresponding modification in the diphtheria-producing organisms themselves. Under the name of pseudo-diphtheria bacilli, of modified diphtheria bacilli, or some such similar name, it has been generally recognised that there are forms which during some stage of their development, at any rate, are morphologically identical with the malignant diphtheria bacillus, although they appear to be devoid of the power of setting up the disease (or they may give rise to it in a very modified form). By many it has been supposed that the attenuated diphtheria bacillus requires the presence of certain special conditions before it can reacquire its virulent form. Leaving out of count differences of food, acidity, the action of air, and other well-known modifying conditions, it is held that it is necessary that it should be allowed to grow on the surface of the mucous membrane of the fauces or even outside the body altogether in the presence of organisms, such as the

streptococcus erysipelas or those streptococci that are found i the throat-affections of scarlet fever, measles, and simila diseases. It is quite possible that the presence of othe organisms may have the same effect, and that the attenuate diphtheria bacillus growing outside the body in the presenc of such organisms may be enabled to acquire such a degre of virulence that it can produce a very grave form of diph theria. Arguing from what usually occurs in the outbreak of diphtheria, in which the first cases, as a rule, are mild, suc cessive cases become gradually more and more severe unti an extremely fatal form attacks susceptible individuals, i would appear that the growth of this bacillus on mucou surfaces along with these streptococci is specially favourabl to the development of the virulent form.

Another series of organisms, in which the special function are closely associated with certain forms of food unde definite temperature conditions, are the light-producin bacteria, about half-a-dozen of which have now been carefull studied, and in which the light-producing function appear to be directly concerned with the special form of metabolis going on in the protoplasm. The photobacterium phosph rescens, for example, grown on ordinary nutrient gelatin to which glucose and maltose have been added, sets up tw fermentations, a colour-forming fermentation and a CO_2 an H_2 fermentation, but only in the presence of oxygen, and a temperatures ranging between 3° and 35° C., as, when th oxygen is cut off, both the ferment production and the phos phorescence are entirely stopped. A second photobacteriu described by Flugge can exercise its characteristic ligh function when supplied with peptone and glucose, but, unlik the preceding species, it is deprived of this power whe maltose is made to replace glucose. Fischer's photobac terium, on the addition of a small quantity of raw sugar, ca produce an intense phosphorescence, but the addition of larger quantity of the same material (3 to 5 per cent.), inte feres with, or altogether stops the phosphorescent activity whilst another form, the photobacterium balticum, is n nearly so sensitive to the presence of a considerable quantit of sugar, as it can live and produce its characteristic light i a medium containing as much as 3 to 5 per cent. of th substance. Other photobacteria do not require the presenc of sugar or of any substance specially rich in carbon an oxygen in order that they may multiply and give off the light, but they have the power, probably through the actio of an enzyme, of liquefying gelatine, whilst in many othe respects they further resemble the ordinary putrefactive ba

teria. Beyrinck, who has made a special study of these bacteria, holds that the formation of light bears no direct relation either to the respiratory processes in the protoplasm of the bacteria or to the growth of the organisms, but he maintains that certain food substances are absolutely necessary for the production of this light, and, although the growth of the colonies may go on perfectly well in the absence of oxygen under certain conditions, the formation of light may be completely stopped. Here, as in the case of other organisms, as soon as the bacteria are grown anaerobically, certain food substances from which oxygen may be easily separated become necessary for the perfection not only of function, but even of growth.

We thus see that the different fermentations and the specific activities of certain of the "disease" organisms are most intimately associated, and that moreover they depend to a very great extent upon the conditions of food supply, accessibility of oxygen, action of light, and other conditions by which they are surrounded; even the kinds of light may affect the powers of the bacteria. Englemann in his study of Ray Lankester's bacterio-purpurin showed that this substance or group of substances in the lower organisms assumes the functions that belong to chlorophyll in the higher plants. If exposed on the microscope stage to the light of the sub-stage spectroscope apparatus, the bacteria forming bacterio-purpurin invariably collect on that part of the slide that is over the ultra-red bands of the spectrum—the portion of the spectrum where the absorption of light by the bacterio-purpurin goes on most rapidly. He finds that this analogy with plants and chlorophyll is still closer from the fact that wherever this grouping takes place, oxygen is set free, and that light is necessary not only for the development of the characteristic colour-producing power, but for the free growth of these organisms. We know also that in the case of the bacillus prodigiosus, oxygen is absolutely necessary for the production of pigment. In the bacillus prodigiosus this colour function may remain entirely in abeyance under certain conditions (such as exposure to a high temperature), the energy of the protoplasm of the organism being diverted to the formation of lactic acid; which substance, however, is formed only in the presence of sugar. These organisms have apparently developed a special power of living aerobically and exposed to the light. Such specially developed functions in them can only be carried on in their entirety when the organisms are placed in what we may call their artificial or specialised environments, and we find further that it is

streptococcus erysipelas or those streptococci that are found in the throat-affections of scarlet fever, measles, and similar diseases. It is quite possible that the presence of other organisms may have the same effect, and that the attenuated diphtheria bacillus growing outside the body in the presence of such organisms may be enabled to acquire such a degree of virulence that it can produce a very grave form of diphtheria. Arguing from what usually occurs in the outbreaks of diphtheria, in which the first cases, as a rule, are mild, successive cases become gradually more and more severe until an extremely fatal form attacks susceptible individuals, it would appear that the growth of this bacillus on mucous surfaces along with these streptococci is specially favourable to the development of the virulent form.

Another series of organisms, in which the special functions are closely associated with certain forms of food under definite temperature conditions, are the light-producing bacteria, about half-a-dozen of which have now been carefully studied, and in which the light-producing function appears to be directly concerned with the special form of metabolism going on in the protoplasm. The photobacterium phosphorescens, for example, grown on ordinary nutrient gelatine to which glucose and maltose have been added, sets up two fermentations, a colour-forming fermentation and a CO_2 and H_2 fermentation, but only in the presence of oxygen, and at temperatures ranging between 3° and 35° C., as, when the oxygen is cut off, both the ferment production and the phosphorescence are entirely stopped. A second photobacterium described by Flügge can exercise its characteristic light function when supplied with peptone and glucose, but, unlike the preceding species, it is deprived of this power when maltose is made to replace glucose. Fischer's photobacterium, on the addition of a small quantity of raw sugar, can produce an intense phosphorescence, but the addition of a larger quantity of the same material (3 to 5 per cent.), interferes with, or altogether stops the phosphorescent activity; whilst another form, the photobacterium balticum, is not nearly so sensitive to the presence of a considerable quantity of sugar, as it can live and produce its characteristic light in a medium containing as much as 3 to 5 per cent. of that substance. Other photobacteria do not require the presence of sugar or of any substance specially rich in carbon and oxygen in order that they may multiply and give off their light, but they have the power, probably through the action of an enzyme, of liquefying gelatine, whilst in many other respects they further resemble the ordinary putrefactive bac-

teria. Beyrinck, who has made a special study of these bacteria, holds that the formation of light bears no direct relation either to the respiratory processes in the protoplasm of the bacteria or to the growth of the organisms, but he maintains that certain food substances are absolutely necessary for the production of this light, and, although the growth of the colonies may go on perfectly well in the absence of oxygen under certain conditions, the formation of light may be completely stopped. Here, as in the case of other organisms, as soon as the bacteria are grown anaerobically, certain food substances from which oxygen may be easily separated become necessary for the perfection not only of function, but even of growth.

We thus see that the different fermentations and the specific activities of certain of the "disease" organisms are most intimately associated, and that moreover they depend to a very great extent upon the conditions of food supply, accessibility of oxygen, action of light, and other conditions by which they are surrounded; even the kinds of light may affect the powers of the bacteria. Englemann in his study of Ray Lankester's bacterio-purpurin showed that this substance or group of substances in the lower organisms assumes the functions that belong to chlorophyll in the higher plants. If exposed on the microscope stage to the light of the sub-stage spectroscope apparatus, the bacteria forming bacterio-purpurin invariably collect on that part of the slide that is over the ultra-red bands of the spectrum—the portion of the spectrum where the absorption of light by the bacterio-purpurin goes on most rapidly. He finds that this analogy with plants and chlorophyll is still closer from the fact that wherever this grouping takes place, oxygen is set free, and that light is necessary not only for the development of the characteristic colour-producing power, but for the free growth of these organisms. We know also that in the case of the bacillus prodigiosus, oxygen is absolutely necessary for the production of pigment. In the bacillus prodigiosus this colour function may remain entirely in abeyance under certain conditions (such as exposure to a high temperature), the energy of the protoplasm of the organism being diverted to the formation of lactic acid; which substance, however, is formed only in the presence of sugar. These organisms have apparently developed a special power of living aerobically and exposed to the light. Such specially developed functions in them can only be carried on in their entirety when the organisms are placed in what we may call their artificial or specialised environments, and we find further that it is

possible by gradually acclimatising the bacillus prodigiosus, for example, to the higher temperature and the diminished supply of oxygen, or by acting upon it with very dilute antiseptic substances through a number of generations, to produce a kind of "race" type as Pasteur and Gessard call it, in which the colour-forming function is replaced by the lactic acid function.

Other bacteria—certain putrefactive bacteria and the typhoid bacillus as shown by Janowski—have their vitality distinctly depressed by the action of light, this action being entirely independent of any oxidation of the food material that may result from the action of the chemical rays; the chemical rays acting directly upon the protoplasms and rendering it incapable, not only of further devolopment but of continuing alive. Janowski, continuing experiments that were first made in this country by Downes and Blunt, took a gelatine tube in which typhoid bacilli had been sown and exposed them to the action of light on a cold winter day; a similar tube inoculated with the same bacillus was wrapped in a layer of black paper, and then in one of white, this also was exposed to the light. Growth in the exposed tube did not occur until the end of five days, whilst in that protected from the light growth commenced in three days. Direct sunlight acting on fluid cultures of the typhoid bacillus was found to kill the organism in as short a time as from four to seven hours; the more diffuse the light the longer the time that was required to kill the organism. Instead of analysing the rays of light by means of a prism, as Englemann and others had done, Janowski made use of solutions of alum, bichromate of potash, Bismark brown, fuchsin, methyl blue, gentian violet, etc., and he found that the yellow and brown solutions filtered out the chemical rays and prevented the action of light on the organisms almost as efficiently as the black and white paper. He found also that the other fluids, fuchsin, methyl blue, and gentian violet, had little more effect in preventing the injurious effect of light on the bacilli than distilled water and alum solution, and he came to the conclusion that the hurtful action of diffused light and direct sun-light is due, in very great measure, to the chemical rays of the solar spectrum.

This question of the action of light, especially on pathogenic-bacteria, is one of almost vital importance to the hygienist; and Duclaux's dictum that fresh air and sunlight are two of the most powerful agents at our command with which to combat the onslaught of certain forms of disease, cannot be too strongly insisted upon. Bacteria, especially those of diseases

such as typhoid, tubercle, and cholera, seek out the dark places for their habitation, and, as the exclusion of light necessitates to a very great extent the exclusion of fresh air, they find in these holes and corners places of rest, whence they may go out to do all the harm of which they are capable.

From the recent experiments on the typhoid bacillus and the pseudo-typhoid bacillus, it is evident, as Chantemesse and Widal pointed out, that one function of the organism —peptonising function—may be held in abeyance by the addition of small quantities of carbolic acid, or, as Hueppe and Wood later insisted, by the addition of traces of glycerine, other functions, even the specific function associated with its power of invading the human subject and forming a specific poison in the body, may, under certain conditions, be imperfectly developed or temporarily held in check. Cassedebat's observations on the different forms of pseudo-typhoid bacillus, which, morphologically identical, differ very considerably in their biological characteristics, give some countenance to such a theory. I might give a large number of other examples of similar modifications, especially if we take up yeasts and the butyric and lactic acid forming organisms, but these are somewhat outside our province, and would be useful only as analogies or examples; and we may leave them out of consideration for the present. On the other hand, this pathogenic power may, under certain conditions, be revived or increased in organisms in which it has not been fully developed, or which have to a certain extent lost what they had previously acquired. Roger and others have pointed out, for example, that the introduction of certain non-pathogenic organisms along with weakened pathogenic organisms rendered the latter more virulent, enabling them to grow and to give rise to the specific products in the body; and, what is more important still, allowing them to acquire a more or less permanent virulence, so that when, after growing along with the non-pathogenic organisms in a host, they are separated as pure cultures and introduced into the fresh host, this time without the assisting organisms, they are still capable of setting up a virulent or a fatal form of the specific disease with which they are associated. This is a most important question and one to which attention is being more and more drawn.

It is difficult to lay down systematically, or with any degree of confidence, the exact advantages that we may expect to accrue from all these observations. We require more detailed and more extensive observation, but I think we are bound to accept the general conclusions that as we

have these modifications of function determined by conditions that in experimental work are completely under control, we may expect, by extending our sphere of operations to the domain of practical hygiene and preventive medicine, so to modify the conditions under which organisms are allowed to remain in our midst, that we may derive from them all the advantages that they are capable of affording us, whilst at the same time we may be able to control or to check their disease-producing powers.

NOTES ON AN EPIDEMIC OF TYPHOID FEVER IN SOUTH-EAST ESSEX IN 1890.

By JOHN C. THRESH, M.B., D.Sc., etc., Chelmsford.

(*Read: April 19th*, 1891.)

In a brief report published last year on the distribution of disease in the county of Essex, I showed that in the southern portion of the county the mortality-rates from diarrhœa and from typhoid fever for the ten years 1879-88 were about twice as great in the registration districts bordering upon the Thames as in the remainder of the county.

The returns for the last two years, 1889-90, show that this disproportionate prevalence of typhoid fever is becoming still more marked. Excluding West Ham, the registration returns for the past five years are as under.

Number of Deaths from Typhoid Fever.

	1886.	1887.	1888.	1889.	1890.	Total.
Rochford	13	18	7	14	22	74
Orsett	5	2	9	9	6	31
Romford	8	5	10	4	15	42
Remainder of county ...	32	37	20	30	29	148

In the next table the death-rates per 10,000 of the population are given.

No. of Deaths and Death-rates from Typhoid Fever in various portions of Essex, including the West Ham Registration District.

	No. of deaths, 1886-90.	Death-rate per 10,000 population.	No. of deaths in 1890.	Death-rate per 10,000 population.
Rochford	74	5.1	22	7.3
Orsett	31	2.8	6	2.6
Romford	42	2.0	15	3.5
Remainder of county ...	148	.8	29	.8

The unfortunate tendency seems to be in the direction of increase rather than of decrease in the typhoid death-rate, more especially in the Rochford district, which occupies the extreme S.E. corner of the county. When the period of

greatest mortality is inquired into it is found to accord with the usual seasonal prevalence. Thus in Rochford in 1889, ten out of the fourteen deaths, and in 1890, sixteen out of the twenty-two deaths, were registered in the last quarter of the respective years.

In the autumn of last year, typhoid fever was unusually prevalent in Southend, a rising watering-place in the Rochford district; and Mr. Morris, the Medical Officer of Health, was requested by the Local Board to report specially on the subject. He did so, and attributed the disease to defects in the sewage and house drainage, confirming, in fact, the reports of his predecessor in office. The Local Board not deeming the evidence conclusive, asked me to investigate the outbreak, and with the consent and aid of the Medical Officer of Health, I undertook to do so.

From inquiries made of the various practitioners in the district, I found that in proportion to the populations, typhoid fever was quite as prevalent in the adjoining villages on the banks of the Thames as in Southend itself, but that it was not nearly so prevalent in the inland villages.

As the Infectious Diseases (Notification) Act had been in force in Southend from the beginning of the year a complete record of the cases was then available. The epidemic of the previous autumn had run its course, and from January to the end of July 1890, only eleven cases had been notified. Towards the end of August, however, the number of cases rapidly increased, the epidemic reaching its height about the end of October, twenty-two cases being notified in the week ending October 23rd. By the end of the year the disease had once more practically disappeared. The number of cases notified during the year was 250, and the number of deaths twelve.

My report of the epidemic was based upon the cases which had occurred up to the end of October, and this report I will very briefly epitomise.

Southend is built upon a patch of brick earth and sand of very unequal thickness, resting upon the London Clay. The older portion of the town is only a few feet above ordnance datum, and this is the district chiefly invaded every year by typhoid. The newer or western portion occupies an almost level tract of land on the cliffs, and having an elevation of about one hundred feet.

In 1881, the population was 7,947, but at the middle of 1890 it was estimated at a little over 12,000. During the summer the place is crowded with visitors and day excursionists, chiefly from the East-end of London and from various parts of Essex.

With the exception of a very few houses, the whole town is supplied with water by a Company, deriving its supply from three bored wells, 906, 876, and 685 feet deep respectively, the borings extending in each case far into the chalk.

Nearly every house is provided with one or more water-closets. Those for the smaller houses are usually in the yards, and a considerable proportion of these are hand-flushed.

There are four systems of sewers. The sewers varying in diameter from nine to fifteen inches, and carrying not only the sewage but nearly all the rainfalls also. The outfalls of two of the sewers are some distance from the shore below the low water level. The other two sewers which drain the old town chiefly, terminate on the beach about 125 yards from the promenade. When the tide is down there is an enormous expanse of mud exposed between these outfalls and the water. This mode of sewage disposal seems to be common to all the villages above referred to in which typhoid is so prevalent.

There was no explosive outbreak at the commencement of the epidemic, though, as the following table shows, there was a somewhat sudden increase in the number of cases notified in the week ending September 4th.

	Cases.		Cases.
Week ending August 7 ...	0	Week ending September 25	7
" " 14 ...	6	" October 2	12
" " 21 ...	3	" " 9	17
" " 28 ...	4	" " 16	15
" September 4 ...	14	" " 23	22
" " 11 ...	15	" " 30	8
" " 18 ...	17	" November 6	15

Of the 156 cases which occurred from the first of January to the end of October, the age and sex has been ascertained in 152.

Age and Sex Distribution.

	Under 1 yr.	1—5 yrs.	5—15 yrs.	15—25 yrs.	25-35 yrs.	35—45 yrs.	over 45 yrs	Totals.
Males	1	9	28	21	13	7	6	85
Females ..	0	1	14	19	12	14	7	67
Totals ...	1	10	42	40	25	21	13	152

From this table we learn that 55 per cent. of the persons

attacked were males, and that 65 per cent. of the patients were over 15 years of age.

The cases had been distributed in 125 houses as under :—

18 houses	with 2 cases in each	...	...	36 cases.
1 house	„ 3 „	...	...	3 „
1 „	„ 12 „	...	...	12 „
105 „	„ 1 „	...	...	105 „
Total ... 125		Total ... 156		

Cause of the Epidemic.—From all I could learn the outbreak differed from those of the preceding autumn only in its more rapid extension. From the first cases were scattered all over the town. This fact, coupled with that of the equal prevalency of the disease in neighbouring parishes, seemed to indicate that neither milk nor water would prove to be the cause. That the milk-supplies were free from blame was easily demonstrated. The houses in which the first sixty-nine cases occurred were supplied by eighteen different milk purveyors, the highest number from any one dealer being eight. The next highest was seven. These firms were also proved to be the two largest dealers in the town.

The water-supply I found was strongly suspected, but for no assignable cause. The other villages in which the disease was so rife were supplied from various sources, the Southend Company supplying Southend and a portion of Southchurch only. Analyses of samples of water taken from the wells and from the mains at various points were carefully examined, and found to accord in all respects with other deep well-water of the district, which I may add are organically of exceptional purity. Dr. M. Tidy, who also examined the water on behalf of the Water Company, certified that it was beyond suspicion. Everything in connection with the waterworks was rigorously examined, but no possible source of contamination was discovered. The origin and distribution of the disease did not suggest any localised defect, neither could any such be found. I came therefore to the conclusion that the water-supply had nothing whatever to do with the epidemic. (The possibility of any other article of food or drink being the cause was also considered, but with negative results.)

My opinion that polluted water was not the cause seems also to be greatly strengthened by a comparison of the course and character of this with other epidemics proved to have originated from defilement of the water-supply. In the following table I have compared the number of cases per 100 houses, the percentage of females and children attacked, etc., in a number of recent epidemics.

Epidemics of Typhoid Fever statistically compared.

Name of District.	Duration of Epidemic.	Reporter.	Population.	No. of Cases investigated	No. of houses att'cked	No. of cases to 100 houses.	D. R. per 100 cases.	P. C. of Females.	P. C. of children under 15.	Most probable cause of outbreak.
Kidderminster ...	Aug.—Oct. 1884	Dr. Parsons	25,500	1,112	690	161	7.5	60	49	Polluted water.
Beverley	July--Aug. 1885	Dr. Page ...	12,000	231	125	185	5.2	59	51	"
Longton	Oct. 1888—Feb. 1889	Mr. Spear .	35,000	155	66	235	19.	61	65	"
Cradley	Dec. 1887—April 1889	Dr. Gresswell	5,284	113	52	217	14.1	—	47	"
Houghton-le-Spring .	April—June 1889	Dr. Page ...	—	275	190	145	9.5	52	52	"
Mountain Ash ...	July—Nov. 1887	Mr. Spear .	12,740	518	265	195	6.	39	29	"
Hitchin	Dec. 1882—Feb. 1884	Mr. Power .	8,500	100alt.	65	154	7.			" chiefly.
St. Albans ...	May—June 1884	Mr. Murphy	11,300	131	90	146				Milk.
Buckingham, Church End	Jan.—Feb. 1888	Dr. Parsons	350	88	30	293	1.1	75	77	Water.
Remainder of Borough ...	Feb.—March 1888	"	2,770	26	19	137	16.	75	46	Defective drains.
Mytholmroyd ...	Autumns of 1887 and 1888	Dr. Page ...	2,270	95	55	173	15.8	55	33	Water and general insanitary condition.
Spennymoor ...	Aug.—Nov. 1888	"		25	19	132	20.0	—	—	Excremental nuisances.
New Clee	March 1887—April 1888	"	17,000	120	55	202	17.5	—	—	Defects of sewerage and house drainage.
Grimsby	Do.	"		140	102	137	18.6	—	—	" "
Southend	Sept.—Oct. 1890	Dr. Thresh	13,000	156	125	125	4.8	45	35	" "

1. *Proportion of Children attacked.*—In epidemics due to polluted water the proportion of children under fifteen attacked is almost invariably high. Thus, at Kidderminster, 49 per cent.; at Beverley, 51 per cent.; at Longton, 65 per cent.; at Cradley, 47 per cent.; at Houghton-le-Spring, 52 per cent.; at Buckingham (Church End), 77 per cent. respectively were attacked. At Mountain Ash only 29 per cent. were affected, but Mr. Spear, who investigated the outbreak there, found a satisfactory reason for this apparent anomaly. At Southend only 35 per cent. of the patients were children under 15.

2. *Proportion of Females to Males.*—In this outbreak the number of females attacked is smaller than in any of the above-mentioned, save at Mountain Ash. But here again Mr. Spear was able to account for the unusual incidence of the disease on the male portion of the community.

3. *The Proportion of Deaths to Cases.*—These I find vary so very widely in various epidemics, of whatever origin, that no conclusions can be safely inferred from the death-rate.

4. *Proportion of Cases to the number of Houses invaded.*—This number also varies considerably, but where water is the cause of the disease the proportion is much higher than I found to prevail at Southend. The observations of Dr. Parsons at Buckingham, as bearing on this point, are particularly interesting. At Church End, a portion of the borough in which the disease first appeared, and was spread by the agency of the specifically polluted water-supply, there were 2.93 cases to each house attacked, whilst in the remainder of the borough, having a different water-supply, and which was invaded later, the proportion was only 1.37 to each house.

5. *Epidemic Period.*—When water becomes specifically polluted an outbreak follows, whatever may be the season of the year, as is evidenced by the dates given in the table. Where outbreaks occur periodically in the autumn in towns having a public water-supply, as at Southend, there is little probability of the epidemic being due to this cause.

The water therefore not being at fault, is it possible that typhoid fever is truly endemic in the district, and that the variation in the extent of the autumnal epidemics is related in some way to the height or the rise and fall of the ground water-level? This would not seem to be the case, for the lower portion of the town is built directly on the London Clay, and it is exactly here where typhoid is most prevalent.

Defects in sewerage and house-drainage alone remain to be considered. My examination of the sewers, of the system of sewage-disposal, and of the house-drainage, led me to the

conclusion that the many defects discovered were adequate to explain the origin of this and previous epidemics. Thus out of 122 houses, in which, up to the time of my examination, cases of typhoid had occurred this year, in fifteen, or 12.5 per cent., cases had occurred once or more before since 1886. Out of thirty-six houses on one sewer outfall no less than eight, or 22.2 per cent., had previously been invaded. Of the forty-nine houses in which fever was known to have occurred in the previous year, in two, cases had occurred in 1888, and in twelve, cases had occurred up to October last year. Whilst taking Southend as a whole only 5.7 per cent. of the houses had been invaded last year up to October, 25 per cent. of the houses in which fever occurred in the previous year had again been attacked. Only defective drains and sewers can account for such facts.

The conclusions at which I arrived as to the cause of the outbreak may be briefly stated :—

1. Climatological conditions especially favourable for the development and spread of the disease.

2. The introduction of cases from without, and the appearance of sporadic cases just at that season when everything was ripe for an outbreak.

3. The small tide-locked, imperfectly ventilated sewers, breathing out at every rise of the tide specifically polluted sewer-air containing the poison in a highly concentrated condition.

4. Flooding of yards, roads, and beach with specifically infected sewage.

5. Direct communication of drains with sewers, bringing the sewer-air into immediate proximity to the houses.

6. The inadequate flushing of the sewers during a very dry September.

7. The absence of any arrangement for isolating fever-patients, so as to prevent infection of the sewers, and the spread of the disease by personal infection.

8. Defects in sanitary administration.

In conclusion, I would only add that the adoption of the Infectious Diseases (Notification) Act in the Southend Urban Sanitary District enabled the course of the epidemic to be easily traced, and that this is one more argument in favour of making this useful Act compulsory. I would, however, go further than this, and urge that the returns should be sent in weekly to the Local Government Board, and that all such as refer to our large towns and to every health-resort should at once be tabulated and published, such other localities from time to time being included if any epidemic prevails therein.

MEASLES: AN EPIDEMIOLOGICAL STUDY.

By A. CAMPBELL MUNRO, M.B., D.Sc.; Medical Officer of Health for the County of Renfrew, lately Medical Officer of Health for South Shields and Jarrow-on-Tyne.

(*Read: May 20th*, 1891.)

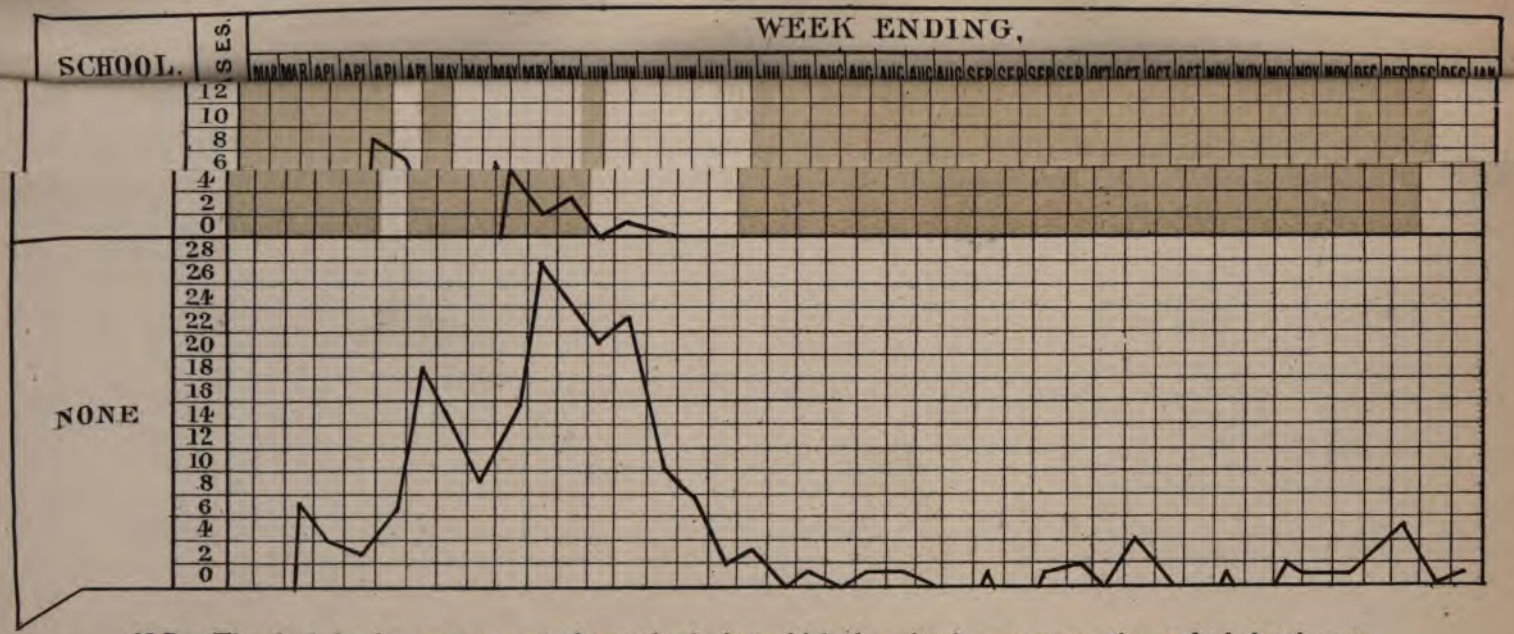
MEASLES may be termed the opprobrium of present-day sanitary science. With the doubtful exception of diphtheria,* it is the only zymotic disease which has not proved amenable to preventive discipline. Cholera has of late years been unknown in England. Small-pox and typhus have almost disappeared. Enteric fever is steadily retreating, as purer sources of water-supply are being resorted to, and filth nuisances are being abolished. The prevalence of scarlet fever is so rapidly diminishing as to suggest in some minds sensations of alarm at the rapidly-increasing mass of the unattacked, and consequently susceptible, element in the population. Even whooping-cough shows a steadily-diminishing death-rate. In the case of measles alone is there an undoubted and steady increase in the mortality-rate. The mean death-rate from measles in England and Wales, which was 377 per million of the population in the decennium 1871-80, has risen to 422 per million over the years 1881-89. I am not aware that any serious attempt has been made to explain this disconcerting circumstance, nor does it appear, judging from the fact that only seven towns in the country, and these inconsiderable in size, have sought notification powers for measles,† that any systematic attempt has been made to remove this stain from the sanitary escutcheon of the country. My attention has been more particularly directed to the matter because of the peculiarly unfortunate experience of Jarrow. There the measles mortality, which stood at the relatively low mean rate of 274 per million of the population in the decennium 1871-80, had risen to a rate of 927 per million over the years 1881-89—and that in the

* I am strongly of opinion that the increase in the diphtheria death-rate is more apparent than real, and is to be attributed in great part, if not entirely, to increased precision in diagnosis.

† Midyear, 1890.

TABLE I.

Prevalence of Measles in connection with School Attendance, Jarrow 1889.



N.B.—The shaded columns represent the weeks during which the schools were open, the unshaded columns, the weeks during which the schools were closed.

TABLE 11.

Death-rate from Measles per Million, Jarrow, 1871–89.

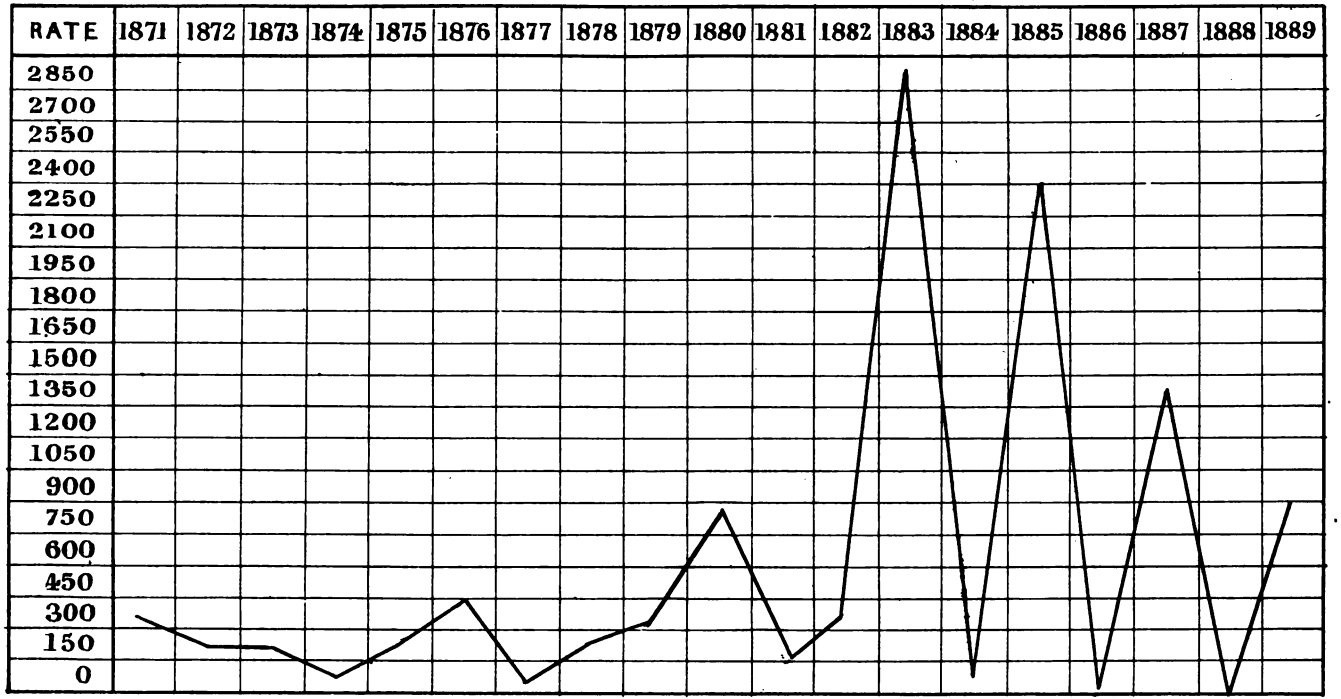
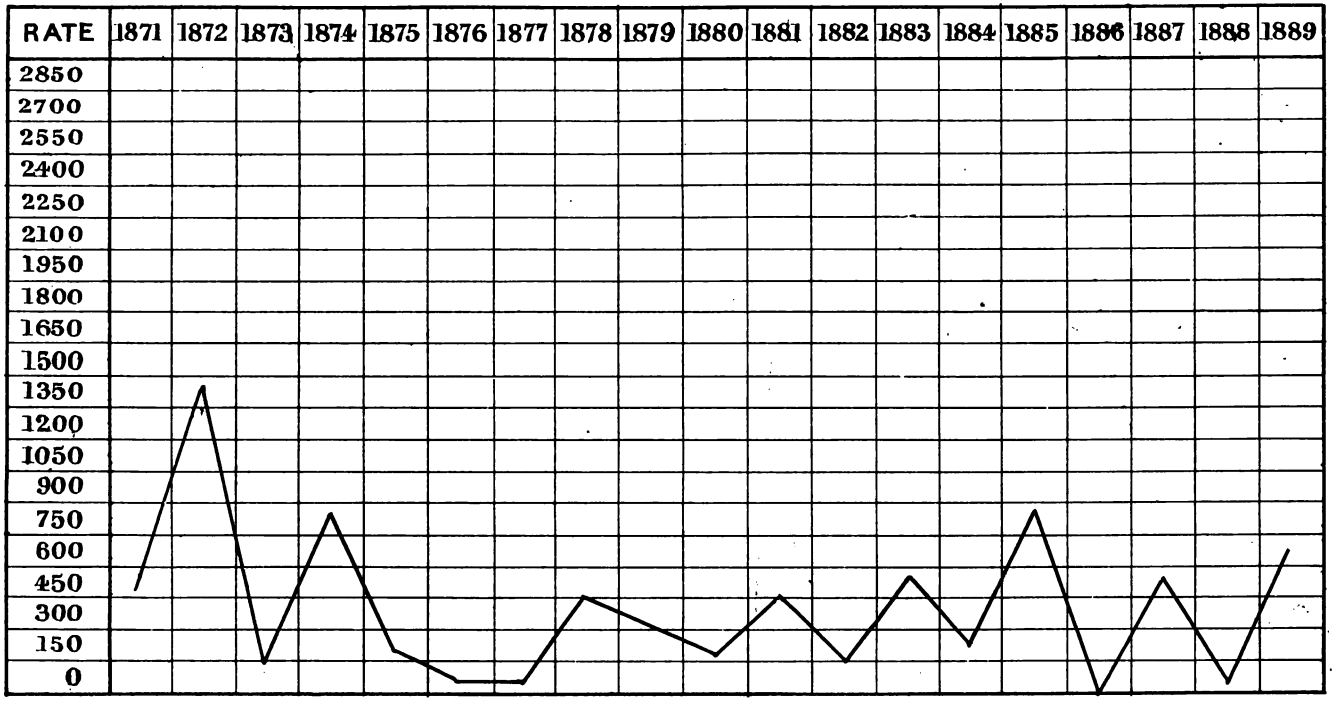


TABLE III.

Death-rate from Measles, per Million, South Shields 1871-89.



face of the most gratifying decline in the mortality-rates from the other zymotic diseases.* So startling an experience has necessarily engaged my most serious consideration, and in the present paper I propose to place upon record the result of my endeavours to elucidate the circumstance, and to obtain an amelioration of so lamentable a concomitant of serious sanitary effort.

I may say at once that my attention has been directed for some time to the question of the existence of any connection between school attendance and the prevalence of measles. It has been urged over and over again that there ought to be no more tendency to the spread of measles amongst children attending school than amongst the same class of children meeting and playing in the streets. It has never been possible for me to accept this view of the matter. As will presently be shown, measles is a disease which spreads chiefly amongst children of Infant School ages. I once took occasion to measure the area upon which a class of children were aggregated in the Infant Department of one of the best Board Schools in my district. The class was engaged in writing an exercise upon slates, and consequently was not so closely massed as it would have been, for instance, at a reading lesson. I found that there were sixty-nine children grouped together within an area measuring fifteen feet by nine! It must further be remarked that at the times when measles tends to become epidemic, *i.e.*, in the early winter and spring months, the ventilation of such schools, dependent almost entirely upon the opening of windows, is reduced to a very small minimum.† Further, according to my experience, the infectivity of measles has singular affinities with that of typhus—its power appears to be in very direct proportion to degree of proximity, and is much diminished by free ventilation. I am unable to believe that there is any equivalence in the risks of infection in the case of children crowded together within a limited area, in persistent contact, their

* Thus, for example, the mean scarlet-fever death-rate over the same periods has fallen from 1,430 to 718 per million.

† In this connection the results of an inquiry with respect to the condition of the air in schools and private dwellings, by Prof. Carnelly (published in the *Transactions* of the Royal Society for 1887), appear to be not without significance. It was found that on an average the carbonic acid present in a litre of the air of schools naturally ventilated was equal to 18.6, as compared with 11.2 in *the night-air of one-roomed houses*, and 7.7 in the night-air of bedrooms in better-class houses. Of bacteria there were present 151, on an average, in the air of the schools, as compared with 58 in the air of the one-roomed houses, and 8.5 in that of better-class houses. In one school the number of bacteria rose to 600 per litre!

respiratory tracts and nervous systems for the time depressed, and consequently in a peculiarly susceptible and receptive condition, as the result of the combined influence of a hot, dry, vitiated atmosphere and mental strain—and the risks in the case of the same children romping in the streets, with the free sweep of the fresh air about them, well-oxygenated blood coursing through their veins, their whole systems intense with vitality and reactive power. It must be remembered, also, that children of infant-school age, when not under the spell of school attendance, tend to linger near their mother's side; at most they play with but the limited circle of their childish acquaintances: how few, then, their chances of exposure to infection among their dozen playmates, as compared with the chances amongst an aggregation of four or five hundred children. In going over the school-attendance register of an Infant School in the course of an epidemic period, it is striking to note the number of children who have been attending school during the initial pre-eruptive, but highly infectious, stage of the disease—not unfrequently, indeed, with the rash out upon them. It is to this risk, peculiar to measles, of children attending school while in a most infectious, although unrecognised, stage of the disease that I have come to attribute the spread of measles in the train of school-attendance. In the case of scarlet fever the risks of infection in the pre-eruptive stage of the disease are fractional as compared with measles. Now, apart from the pressure of school attendance, children in this initial stage, feeling more or less unwell, would in all probability linger by the fireside—especially at those ungenial seasons of the year at which the disease tends to become epidemic—rather than rush into games of romps in the streets.

Under the influence of these considerations it occurred to me to inquire whether the school-attendance returns, over a series of years, threw any light upon the causation of this ill-omened increase in the prevalence of measles in Jarrow in recent years. I obtained the returns of the average school-attendance in each public elementary school in Jarrow from 1870 to 1888, and tabulated the results, as per thousand of the population, year by year. These results were very striking. I found that the school-attendance rate, which was as low as 52 per thousand in 1871, arose by a tremendous leap from 68 in 1874 to 162 in 1882—the year which preceded the first great epidemic of the present decennium; while the mean number of children in average attendance at school rose from 95 per thousand of the population in the decennium 1871-80, to 164 over the years 1881-88. Dr.

Ransome's theory* of the production of epidemics is that the chief determining cause is the degree of proximity of susceptible persons. How great an increase in the aggregation of persons at the most susceptible ages within the walls of our elementary schools the above figures represent! Upon Dr. Ransome's theory, these figures fairly account for the fierce epidemic outbursts of the disease, and the greatly increased mortality, which have characterised the present decade as compared with the last. I have made diligent inquiry after any other influence to which, in any degree, the increased prevalence of measles in Jarrow in recent years might be attributed, but I have been unable to obtain a suggestion of any other efficient cause than that of the increased aggregation of susceptible material in our public elementary schools; and what holds good in Jarrow I believe to be valid in explanation of the increase in the prevalence of measles in England and Wales generally. Jarrow is a purely working-class town, and the aggregation of children in the public elementary schools is excessive. In towns with a larger proportion of people of better social position, the proportion of children attending the public elementary schools will fall, and with it the tendency to excessive epidemicity, *ceteris paribus*, diminishes. Such, I believe, is the explanation of the fact that, in the town of South Shields, which is contiguous with Jarrow, measles has shown no such tendency towards excessive epidemicity as Jarrow, in the present decade. [See Diagram III.] South Shields is a "better-class" town as compared with Jarrow, and there is not the same aggregation of children in the public elementary schools. There is reason to believe, further, that the School Board of South Shields is not so exacting in the matter of school-attendance as the School Board of Jarrow. At any rate, the school-attendance rate in South Shields was 96 per thousand in 1874, and rose only to 122 in 1882—an increase of only 26 per thousand, as compared with an increase of 94 per thousand in the case of Jarrow on a comparison of the same years. The *mean* "average school attendance" in the first eight years of the present decennium has been only 133 per thousand of the population in South Shields, as compared with 164 in Jarrow. My theory is that in the case of South Shields the increase in the intelligence of the people and the increase in their appreciation of the dangers of infectious disease have sufficed to neutralise the risks involved in the relatively small increase in the school attend-

* *Epidemic Cycles*, by Arthur Ransome, M.A., M.D.; *Proceedings Lit. and Phil. Soc. Manch.*, vol. xix, No. 8.

ance; while the huge rise in the school-attendance rate in Jarrow has far outstripped the increase in the average intelligence of the people in matters pertaining to infectious disease.

So far we have been dealing with an aspect of the case which is largely speculative. In the course of the year 1889 I had an opportunity of applying the touchstone of experimental research to the question. In 1887, when, in Jarrow, we had only got the length of a defective system of voluntary notification of cases of measles, I made an effort to arrest the progress of an epidemic of measles by securing the closure of the Infant Department of two of the public Elementary Schools of the town.* The attempt was but partially successful. Experience has shown me that, in the absence of a complete system of notification, any attempt to control the spread of measles by resort to school closure must in a town be more or less blind, clumsy, and ineffective. In 1889, when our next epidemic of measles occurred, we were much more favourably situated. We had by that time obtained powers for the compulsory notification of measles. Realising that we had now a unique opportunity of studying the epidemiology of measles, a close record was kept of all the circumstances connected with each case, which was notified from the commencement of the epidemic. The one dominant circumstance which that record reveals is the relation between school-attendance and the extension of measles. There was no relation whatever discovered between milk-supply and the spread of the disease. I endeavoured, by means of spot-maps, week by week, to trace any evidence of any considerable extension by ordinary intercourse along a street, or in particular localities, but in vain—with one exception, when a sudden outburst occurred in a previously unattacked (out-lying) street (Prince Consort Road), and then the children attacked were almost all in attendance at the Monkton School—a school which had previously been free from the disease. Diagram I embraces all the facts in connection with the relation between school-attendance and the prevalence of measles in Jarrow in 1889. It covers the whole duration of that prevalence in a series of

* Previous experience had shown me that measles, in Jarrow at least, is a disease of infant-school age, and that the attendance in connection with the senior departments of the elementary schools is not appreciatively affected during an epidemic of measles. In the course of the whole epidemic of 1889, presently to be referred to, only ten children in attendance on the senior departments of the elementary schools of the town were attacked. On an analysis of the cases occurring, I find that, excluding the few adults who suffered from the disease, the *mean* age of those attacked was 4.2 years.

weekly periods. The weeks darkly shaded are those during which the schools (specified in the first column) were open, the unshaded weekly periods are those during which the schools remained closed. The zig-zag lines represent the prevalence of measles amongst the children usually in attendance at the different schools. The lowest line, however, represents the prevalence of measles amongst children under school age. To avoid any confusion of the issue, only the first cases occurring in households are taken cognisance of, it being assumed that subsequent cases occurring in the same household have in all probability derived their infection from the first. It must further be explained that the date of the eruption is adopted for the purposes of this table as the date of the "occurrence" of the case, as it affords a fixed point of departure which the "onset of the illness", as observed by the friends, would not do; frequently, for instance, no sign of illness was observed until the rash had appeared, while in other cases the friends fixed the onset of the illness at some weeks before the appearance of the rash. This arrangement adopted, of weekly periods, has the advantage of fitting in with the duration of the incubative period; in my experience the usual date of the appearance of the rash may be set down at fourteen days after the date of the infection, although in some exceptional cases it appeared to be a day or two earlier or a day or two later of appearing. Finally, it may be admitted that, in spite of compulsory notification, I did not hear of every case of measles which occurred in the course of the epidemic; I may be permitted to say, however, that I spared no pains to trace the existence of unnotified cases. Let me again explain that the average number of children attending school was over 1,000 in the case of the Grange, Catholic, and Dunn Street Schools, 700 at the Ellison School, 400 at St. Peter's School, 300 at the Wesleyan School, 130 at Monkton. Equipped with these explanations we shall find the table a most interesting and instructive study.

While there had occurred in the earlier months of the year a few sporadic cases of the disease, in the first week in April I was brought face to face with a most remarkable outbreak and extension of the disease: in the week ending April 5th there occurred no fewer than thirty-six cases of the disease, being at the rate of more than one per thousand of the population; in no single previous week had there been more than two cases notified. But a still more striking feature characterised the outbreak; of the thirty-six children attacked twenty-nine were attending school, and *of these twenty-nine*

no fewer than twenty-eight were attending the Infant Department of one particular school—the Grange Board School. In this outbreak twenty-six cases in attendance at this school were notified before any case was reported from any other school in the town. There could be little doubt that some specially infective influence attached to attendance at this school. The children attacked were scattered, in residence, all over the town; they belonged to different social grades, and, in addition, being infants, were not likely to come into contact out of school hours. What the specially infective influence was, in the first instance at least, I was able pretty clearly to make out, as the result of subsequent inquiry. A fortnight before, a child, with the rash of measles upon her, had been unwittingly sent to school. When the rash was discovered the teacher at once sent her home; but as measles is undoubtedly infective for a couple of days before the rash comes out, the child had evidently been in school-attendance while in an infectious condition for three days. My course I considered clear. The Sanitary Committee met on the evening of April 4th. I advised the Committee to make an order closing the Infant Department of the Grange School forthwith. This was done, but the order was not valid until confirmed by the Council, which did not meet for six days, in the course of which much harm might be done. The examination of the school by H.M. Inspector was fixed for the following week. Next morning, with the clerk to the School Board, I went to see H.M. Inspector with reference to the matter; he readily consented to recommend the Education Department to give the same grant to the school as it had received the year before—which happened to be the highest possible — and without examination, so as to place no obstacle in the way of the closure of the school. Accordingly the school was closed the same day at mid-day. The incubation period of measles—the period which elapses after the reception of the infection before the disease becomes manifest through the characteristic rash—may, as I have said, be set down at fourteen days. In the week following the termination of these fourteen days only one new case was notified amongst the children who usually attended the Grange School, and the epidemic, so far as this school was concerned, was at at end, only occasional sporadic cases occurring at intervals of many weeks during the further course of the disease in its epidemic form in the town.

A second outburst of the disease in connection with this

school occurred in the month of November; to that I shall allude further on.

Let us now take the equally significant case of the Catholic School. The disease did not make its appearance among the children attending this school until the week ending May 3. It sprang into virulent activity in the course of the week ending May 24th, when there were twenty-two cases notified amongst the children attending the school; in the course of the next week there were fifteen cases. Early in this week I secured the closure of this school without a formal order. At the end of the probationary fortnight, cases occurred amongst children ordinarily in attendance at this school at the rate of only one per week. After the expiry of a period of three weeks the disease had entirely disappeared from amongst the children who had been in the habit of attending this school. We had a parallel experience in the case of the other schools, with the exception of Dunn Street Board School. There, it will be observed, there occurred six cases in the *third* week from the closure of the school. But of these cases five occurred in the first two days of the week; and if we may accept it that the incubation period is sometimes extended by a couple of days, these cases prove no real exception to the general experience. Acting on my advice, the Sanitary Committee made an order anticipating the period of the annual holiday, and closed all the schools in the town for a period of five weeks from the 7th of June; simultaneously the managers of all Sunday Schools within the borough were requested to close the infant departments of their schools. A glance at the diagram will show that within a month the epidemic had become practically extinct; in the week ending July 26th there were no cases of measles reported within the borough. It may be urged that the epidemic had then simply burned itself out, that it would have died out at that time in any case. There is strong evidence to the contrary, however, in the experience of the contiguous town of South Shields. There we were without compulsory notification, and we were consequently unprovided with the data, without which we should not have been justified in resorting to the strong measure of school closure; the schools broke up for their summer holidays at different dates, as their managers chose. The epidemic in Shields arose and spread practically simultaneously with that in Jarrow, but it only attained its height (so far as we can judge from the number of deaths) in the month of July, and had but little diminished in intensity for three months thereafter:—

Deaths from Measles, 1889.

	Jan.	Feb.	Mar.	Apl.	May.	June.	July	Aug.	Sept.	Oct.	Nov.	Dec.
South Shields...	0	1	1	0	2	2	15	6	9	9	2	1
Jarrow ...	0	0	0	4	3	15	2	1	0	1	1	1

It will be seen that, as indicated by the deaths, the epidemic in Jarrow was in full swing in the month of June, the month in which all the schools were closed together. The later experience of the Grange and Dunn Street Schools in Jarrow shows pretty clearly that the outburst of the earlier months had by no means exhausted the available material; for it will be seen that a recrudescence of the disease in its epidemic form occurred in connection with the Dunn Street School in September and October; while there was a vigorous outburst of the disease in connection with the Grange School in the month of November. I may be asked, especially in connection with the latter case, two questions: Why I did not again resort to school closure, and in what way I explain the sudden termination of the outburst without resort to school closure? My reply to the first question is twofold: first, that by the time I became aware of the seriousness of the outbreak, in the week ending Nov. 22nd, before I could have obtained an order for the closure of the school there would have been a week's loss of time at a most critical period; and, second, that the disease having practically disappeared from the town elsewhere, I was able to devote an amount of personal attention to that school which was equivalent to school closure for the effective purpose of excluding suspected children—children suffering from symptoms which might be premonitory of measles—from school attendance. Under my supervision the teachers, chief and subordinate, warned by the unfortunate experience of the earlier months, and with the fear of school closure before their eyes, were on the *qui vive* to observe any coryzal symptoms amongst the children attending school, and to send the subjects home for a few days' quarantine. Were it possible to devote, and obtain, as much individualised attention to every school amongst the children attending which measles had broken out, there would be no need to resort to school closure in order to cope with the disease. To the second question I venture to reply that, in all probability, the outburst was arrested through the personal supervision I was able to bestow upon the matter of school attendance, backed up by effective aid from the teachers.

In studying this diagram in the light of what has been

already observed, it is important to note that the outbursts in connection with the different schools were not synchronous, as they practically would have been had they simply been parts of a general epidemic. The small epidemics (if I may so call them) in connection with the different schools ran independent courses. Thus, we have the first outbreak in connection with the Grange School running and terminating its course before, for instance, the disease makes its appearance in the train of the attendance at the St. Peter's, Catholic, Wesleyan, or Monkton Schools. We have the outburst starting in the case of St. Peter's and Catholic Schools when it had practically disappeared in the case of the Grange, Dunn Street, and Ellison Schools, and still more clearly we see the second little epidemic in connection with the Grange School arising and running its course, while no single case occurs amongst children attending any other school in the town. Another point of interest brought out in the diagram is that the highest rate of prevalence amongst children under school age is *sequent* to the highest points in the curves of the school-attending cases.

Leaving, for the time, the question of measles in its relation to school-attendance, I should like to refer to some other points of interest in connection with the epidemiology of measles which have been placed in relief by our experience in Jarrow. A reference to the diagram—Table II—brings out, in the most astonishing manner, the biennial epidemicity of measles in Jarrow in the present decennium. In 1883 the measles death-rate was 2,929 per million; in 1884, 103; in 1885, 2,400; in 1886, 33; in 1887, 1,443; in 1888, 0; in 1889, 903;* and with such an experience I am justified in anticipating at least a tendency to epidemicity in 1891. The question of epidemic cycles is one of great interest; the *fact* has probably never been more clearly demonstrated than in the experience of Jarrow. In the case of South Shields the same fact is illustrated, although upon a smaller scale—there, measles has been epidemic in 1881, 1883, 1885, 1887, and 1889. Various theories in explanation of the fact of epidemic cycles have been advanced, some of them of the most mystical and transcendental character, few of them based upon any substantial foundation of fact. Dr. Farr's hypothesis, based more particularly upon our experience of rinderpest, that the poison of the disease is itself so diluted by successive transmissions through the bodies of its victims, that it becomes gradually less and less able to propagate the complaint, accounts for some of the facts of the case, but not

* There was no death from measles in 1890.

all; it requires as Dr. Ransome has pointed out, the further assumption that after a period of latency, the poison gathers strength enough again to attain an epidemic violence. I believe that no hypothesis which refers this tendency to epidemic cycularity to something in the nature and life history of the contagion, will satisfy the requirements of the case. Taking the case of the disease with which we are immediately concerned, measles, we find, as the result of Dr. Ransome's examination of the Swedish tables of mortality over a period of a hundred years, that in that country measles has shown a distinct tendency towards a septennial epidemicity. In Jarrow, as we have seen, the cycle is biennial. Yet the poison is the same in both cases. I am satisfied that the real explanation of the occurrence of epidemic cycles is that formulated by Dr. Ransome, "that these disorders can only become epidemic when the proximity between susceptible persons becomes sufficiently close for the infection to pass freely from one to the other". Or, put in another form, that the tendency towards cycular epidemicity arises out of external conditions, and is not due to anything in the essence of the *materies morbi;* and that the main external condition concerned is that of proximity of susceptible persons. Thus, in a sparsely-populated country like Sweden the epidemic cycle is elongated; with a closely-aggregated population of children at the most susceptible ages, as in Jarrow, the cycle is shortened. The circumstances of the Jarrow case, as above detailed, fit in extremely well with Dr. Ransome's hypothesis. There are, doubtless, other subsidiary modifying factors, as, for instance, varying social conditions (although these are partly included in Dr. Ransome's definition), seasonal influences, etc.

As the result of considerable research I am able to affirm that, under presently-existing conditions in England, the natural tendency of measles is to a biennial epidemicity. The measles death-rate of England and Wales, over the years 1871-89, exhibits a distinct, although much subdued, biennial excess; here, however, an epidemic prevalence of the disease in one part of the country, in any year, is masked by a diminished epidemicity in another part. The real state of the case is revealed when the circumstances of individual districts are inquired into. Jarrow has already been dealt with. In the case of South Shields we have a like experience, although upon a smaller scale. The measles epidemic-curve never rises to anything like the same height as in Jarrow. Yet here, too, the biennial cycle stands revealed: 1881, 1883, 1885, 1887, and 1889 were years of epidemic

prevalence of measles in South Shields. I have been favoured with returns of the measles deaths in their districts over a series of years, by the health officers of important towns scattered over the country; from these returns I learn the following facts. In the case of Newcastle-on-Tyne the prevalence of measles has risen to epidemicity, as in Jarrow, in the years 1883, 1885, 1887, 1889. In Sunderland it was epidemic, although on a much reduced scale, in the years 1874, 1876, 1878, 1880, 1882. As the result of the operation of some factor which I am unable to determine, it missed the year 1884; but in 1885 it again assumed an epidemic form, and the biennial cycle is again continued in the years 1887-89. In Edinburgh there is a distinct tendency towards a biennial cycle, but it is modified by some influence or influences which tend to shorten the cycle. This is shown very clearly in a diagrammatic table appended to a most interesting paper by Dr. Harvey Littlejohn, entitled "Ten Years of Compulsory Notification of Infectious Diseases in Edinburgh".* Beginning with the year 1880 this diagram shows a tremendous epidemic outburst in the later spring, attaining a maximum of 1,150 notified cases per month. In 1881 the disease did not become epidemic, although cases were notified in every month of the year. Again, in the later spring of 1882 came another huge conflagration, no fewer than 1,210 cases being reported during the month of highest prevalence. Following a strictly biennial course, the next epidemic was due in the spring of 1884; but here some new influence evidently came into play, favourable to the rapid spread of the disease, and the epidemic expected in 1884 was anticipated by an epidemic prevalence of the disease beginning in November-December 1883, but continued in a diminished degree over the spring of 1884. We look forward to the next epidemic as due in the spring of 1886; and sure enough it comes then with unexampled fury. Starting with a considerably increased prevalence in the month of February, it rises to a maximum of 1,480 notified cases in the month of May. We look for the next epidemic in the spring of 1888, and our expectation is fulfilled, although the date is slightly anticipated, the highest prevalence of the disease being in the month of January, with over 1400 notified cases; the most of the epidemic curve, however, falls in the spring of 1888. But in the case of the next epidemic we find the biennial cycle shortening: for the disease has assumed epidemic proportions in November 1889, and risen to 1460 notified cases in

* Oliver and Boyd, Edinburgh.

the month of December; its epidemic prevalence is continued over the spring of 1890. I think it highly probable that the main factor tending to shorten the epidemic cycle of measles in Edinburgh, and to anticipate, by a few months, the biennial spring epidemic (which we look for in the North-east of England) is the influx into Edinburgh in the course of the month of October, for educational purposes, of a considerable amount of fresh and susceptible material from rural districts.

Passing to the far south, we find in the case of Portsmouth the most typical illustration of the biennial measles cycle. As in the case of the northern towns, we find the smaller elevations representing in our diagrams the measles prevalences of the last decennium, translated in the present into mountain peaks. But over the whole of the two decades every second year is characterised by an increased prevalence of measles, rising in the years 1882, 1884, and 1886, into violent epidemicity. It is interesting to note in passing, that these epidemic prevalences attach to the even-number years of the series, thus masking in the mean death-rate of England and Wales the epidemic mortalities characterising the odd number years in the north of England. The Bristol measles-mortality curve is most erratic. Taking the whole period 1871-89, it shows a distinct tendency towards biennial epidemicity, but this is lost or obscured over the years 1879-83; and over the whole period the intermediate years do not show that almost entire absence of measles prevalence which characterises the towns already referred to. Measles was excessively prevalent in Bristol in the years 1873, 1875, 1877, but the drop in the intervening years was relatively small. The curve does not rise materially in 1879, while it is consecutively high in 1880, and 1881, and consecutively low in 1882, 1883, 1884. It returns to a rate of biennial epidemicity in 1885, 1887, and 1889. Coming northwards again to Bradford, we find in the earlier part of the period 1871-89, a distinct biennial epidemicity in the case of the years, 1872, 1874, 1876. After that time the biennial ascent is blurred, or lost, and the curve seems to follow no particular law of periodicity. In the case of Salford, the last of the series of towns I have information from, over the whole period 1871-89, there is evidence of a tendency towards biennial epidemicity only in the earlier and later parts of the period; in 1871 and 1873 measles appears to have been epidemic; it does not again rise in 1875, and it pursues a quite erratic course until, in the later part of the period, it regains its biennial epidemicity in the years 1885, 1887, 1889.

Permit me to summarise the provisional conclusions at which I have arrived, as the result of the consideration of the above detailed circumstances, and others which it would be impracticable to include in a paper of reasonable length. First, that the chief factor in the production of the increased death-rate from measles over England and Wales, and more especially in particular districts, in the present decennium, is, in all probability, the greatly increased aggregation of susceptible material in our elementary schools, and that the most usual source of infection is probably the attendance at school of children in the pre-eruptive but infective stage of the disease.

Secondly, that the control of the spread of measles in urban districts is by no means, as has been assumed, a matter for despair;* that, given compulsory notification of all cases of measles, a careful and prompt return to the Medical Officer of Health of the circumstances of each case as it occurs—more particularly of the school attendance, and the immediate closure of every infant school amongst the school attendance of which, say, from six to ten cases (assuming an average-sized urban school) have occurred in the course of a week—the spread of measles is well within control. No experiment has yet been made under circumstances quite so favourable. In preparing for another epidemic outburst in Jarrow, the first point I should endeavour to secure would be the endowment of the Sanitary Committee (which could be called together at a few hours' notice) with plenary

* Many health-officers entertain pronounced opinions as to the inutility of compulsory notification of cases of measles; our experience in Jarrow furnishes strong presumptive evidence to the contrary. The first great epidemic of the decade occurred in 1883. At that time a few cases were voluntarily notified, but notification was not asked for by the Corporation, and no greater attention was paid to the matter than is customary in other towns. There occurred in the course of that epidemic 82 deaths from measles (I am not, of course, in a position to state the number of cases); the measles death-rate was 2,929 per million. The next epidemic visitation was in 1885; when the disease had attained epidemic proportions, the medical men of the town were requested by the Corporation to notify cases, and a tardy effort was made by the health-department to cope with the disease. The number of deaths this year was 72, the death-rate, 2,400. In 1887 the disease again showed signs of spread; the Sanitary Authority at once invited notifications, for which they offered a half-crown fee; school closure was resorted to in two instances, although somewhat late in the day; and, generally, a serious effort was made to arrest the course of the disease; this year the death-rate fell to 1,443. Before the next epidemic, of 1889, we had scheduled measles as one of the diseases compulsorily notifiable under the Jarrow Improvement Act. The inevitable outburst came, was energetically dealt with, and the death-rate fell to 903. It is difficult to explain this steady and uniform decline in the extent of the measles epidemic in Jarrow upon any other ground than the utility of the notification system in dealing with measles.

powers for the immediate closure of schools: I believe, after our previous experience, I should have no difficulty in persuading the Committee to exercise their powers as promptly as above suggested. The senior departments of the public schools, attended by children who have passed the most susceptible ages, could generally be efficiently dealt with, without closure, by effective supervision and the retention from school of children from infected houses. When an epidemic of measles threatens, the managers of Sunday schools should be requested, by a resolution of the Committee, which should be communicated to the press, not to receive in school attendance children under eight years of age, or children from infected houses.

Thirdly, I think it is clear that measles shows a distinct tendency to a biennial cycle of epidemicity under existing conditions in England.

Fourthly, I may be allowed to remark, although the facts are not set forth above, that our experience in Jarrow coincides with that already recorded in other places; that measles tends to become epidemic at two seasons of the year, one period having its maximum in April and May, the other in November, December, and January. I remark, as throwing some light upon this phenomenon, that I find, in my own district at any rate, that the highest point in the death-rate from acute pulmonary diseases is reached in April and December; it may be assumed that at these periods the air-passages are in their most irritable and receptive condition.

Permit me to insist, in conclusion, that I do not pretend to have *demonstrated* these propositions; that, especially with respect to the question of the utility of school closure, was impossible. It is open to anyone to say that in each case the outbreak was already on the wane when the school was closed, and would have become extinct in any case; and it is, of course, impossible to prove a negative. This argument, although a somewhat hazardous one to apply, has a certain force as the result of our experience in connection with the decline of many epidemics; but when used in relation to isolated bits of a general epidemic, each dependent, perhaps, upon the accident of the attendance or non-attendance at school of a single infectious child, the hazards of the argument outweigh its utility. What I do claim with respect to this particular matter is, that, for the first time, a careful record has been kept of the school attendance relations of every case of measles notified in a district in which the compulsory notification was in operation; that

the circumstances attendant upon certain instances of school closure have been carefully recorded; that those records afford a strong presumption in favour of the judicious application of school closure; and that if the experiment of school closure is carried out under similar circumstances of careful observation, with a like favourable sequel, in, say, half-a-dozen instances, a result will be arrived at which will, for all practical purposes, be the equivalent of mathematical demonstration.

ON THE SPREAD OF INFLUENZA BY CONTAGION.

By R. SISLEY, M.D.

(*Read: May 20th*, 1891.)

Sir James Moore, in a paper which he recently read before the Epidemiological Society, strongly pointed out the injurious effect of attaching too much importance to "authority" in matters of science. I have lately been much struck by the truth of this observation.

Until the occurrence of the epidemic of influenza in 1889-90 very few living physicians knew from personal observation anything about the disease or its method of spread. They had, therefore, to rely on the teaching of the past, and I think many of them attached almost implicit reliance on the statements of Sir Thomas Watson, many of whose observations they have been able to verify in the case of other diseases, and whose writings possess a charm which is certainly absent in current medical text-books.

On the method of the spread of influenza Watson taught that contagion played but a small part. "I have remarked," he says, "that Cullen makes this species of catarrh to proceed from contagion. But the visitation is a great deal to sudden and too widely spread to be capable of explanation in that way. The occurrence of epidemic catarrh, as well as of most other epidemics, is unquestionably connected with some particular state or contamination of the atmosphere. . . ."*

This teaching was, I think, generally accepted, and Watson's classical description of the disease, as it occurred in 1837, was extensively quoted. But the epidemic of 1889-90 was by no means a sudden visitation. There were isolated cases in different parts of the town long before there was any general infection of the population. Many of the first sufferers were seized immediately after their return from Paris, and some of the first cases I saw were patients from hotels where foreign travellers had been staying.

* *Lectures on the Principles and Practice of Physic*, vol. ii, p. 41. Lond., 1853.

Early in the epidemic facts came to my notice which pointed to contagion as an important means by which the disease was spread. The infection in the earliest case recorded in England* was, I believe, contracted at a Christmas party, and I felt convinced that I took the disease from this patient. Early in January, whilst I was out of town trying to get over the effects of the malady, I was called to see a lady who had a recurrence of the disease. A young lady who was staying with her (and who was much in her company both by day and night) soon took the disease, and in her turn, as it seemed to me, communicated it to a servant who was with her in a house to which she afterwards went. These observations are not in themselves striking, and I only quote them because they inclined me to the view that the disease was contagious; and that idea, together with a knowledge of the fact that influenza in London occurred at first in isolated centres, led me to take an interest in the general spread of the disorder through England, and I became doubtful about the truth of the conclusions arrived at by Watson as to the method of the spread of the malady. I look forward to the report of the Local Government Board, which will, doubtless, be of great use in showing (1) the date of the first appearance of the disease in different parts of the country; (2) how far the first cases occurred in people who had come from infected places; (3) how long a time elapsed between the first appearance of the disease and the general infection of the population. Until the report is published we must content ourselves with the information at present at our disposal, and, fortunately, the newspapers both of town and of country districts give many facts.

Without relying too confidently on the absolute accuracy of this information, and making allowance for possible errors, we are able to get a general view of the progress of the epidemic.

Edinburgh it seems was affected about the same time as London and Colchester. Canterbury and Portsmouth suffered early in January. The disease was spreading in Devonshire and Monmouth during the first week in February, and was raging in Wales as late as March.

At Chelmsford, Durham, and Bury, isolated cases occurred before there was a general infection of the population, and at the latter place people living in the town were affected before the inhabitants of the surrounding villages suffered.

* *Lancet*, Jan. 4, 1890.

In Nottingham and Birmingham the railway servants were the people first affected.

It would be tedious and useless to give a full analysis of the information given in a collection of many hundreds of newspaper cuttings; but a careful consideration of the material at my disposal convinces me that towns were generally affected earlier than country districts, and the more remote parts of the country later than places near London; that in many towns there is distinct evidence that the disease first occurred in isolated centres; that there is no evidence of the sudden seizure of a large number of people without the previous existence of isolated cases of the disease; finally, that where there was little human intercourse, villages and country districts directly between large towns often escaped infection till some time after the towns were affected.

The last-mentioned observation disproves the theory that the infection is entirely wind-borne. A valuable observation bearing on this matter was made during the epidemic of 1782, and was recorded by Dr. Gray on the authority of Dr. Ruston, of Exeter. Writing of the spread of the disease, he says: "At Dartmouth it began much sooner, and at Tinmouth, which lies between both, much later, than at Exeter."*

I do not think that the importance of such facts can be overlooked in a consideration of the method of spread of the disease; *i.e.*, as to whether personal contact or what is called aerial infection forms the chief means of conveyance.

The idea that contagion plays a great part in the spread of the disorder is a very old one, and as early as 1743, when Rome was affected, the Pope ordered a land quarantine to check its spread. In the same year, at Messina, a ship suspected of being the means of conveying the disease was burnt, and all letters from foreign parts were perfumed.†

The records of the old English medical writers are full of facts pointing to the spread of influenza by contagion.

In 1782, according to the evidence of Dr. Clark, the disease was imported to Shields from London by a ship.‡ In the same year the first two patients who suffered from the disorder at Norwich were people who had lately arrived from London, where it was then raging.

Dr. Haygarth, of Chester, gave many similar examples.

* *Medical Communications*, vol. i, p. 61, *note*. London, 1784.
† *Gentleman's Magazine*, vol. xiii.
‡ *Medical Communications*, vol. i, p. 63, *note*. London, 1784.

In answer to a question as to the introduction of the disease, he wrote:

"1. That the first patient who had the disease in Frodsham was seized with it as he was returning thither from Manchester. 2. That at Malpas, the first patient was the landlady of the inn and her family, a week sooner than any other patient in the town. 3. That the first person who had the distemper in Middlewich brought it from Liverpool. 4. That the first person affected with the influenza at Mold had been at Chester a few days before, in a family ill of the distemper. 5. That a gentleman arrived at Oswestry ill of the influenza before the inhabitants were attacked. 6. That at Tarporley the first person seized was a postillion who had driven a chaise thither from Warrington, where the distemper had previously appeared. 7. That at Wrexham the first patient came from Chester, and the second from Shrewsbury."*

In the epidemic of 1803 similar cases were reported from Exeter, Cirencester, and other places, and there was evidence that the disease was imported into the Isle of Man by a Liverpool packet.

During the late epidemic, I think sufficient importance was not attached to the contagious nature of the disease, and many medical men thought that direct infection played quite a subordinate part in the spread of the malady, or even that the disorder was not contagious at all. Thus, the report issued by the Local Government Board for Ireland contains the following passage:—

"On the subject of the origin and mode of extension of the epidemic in Ireland, the views expressed by medical officers differ, but that a general consensus of opinion to the effect that the disease was of a miasmatic character, that it was air-borne, that it was preceded and accompanied by high temperature and moist atmosphere. A small number of observers consider it infectious and contagious, but the great majority were of opinion that it was not."

Dr. William Squire, in an article on "The Infection of Epidemic Influenza",† after reviewing the evidence he gave, came to the conclusion that "the balance seems rather to be against direct infection as a frequent or potent cause of the spread of influenza".

* *Annals of Influenza or Epidemic Catarrhal Fever in Great Britain from* 1510 *to* 1837. Prepared and edited by Theophilus Thompson, M.D., F.R.S. Lond., 1852. I have been unable to verify this quotation by referring to Dr. Haygarth's original paper, of which I have not been fortunate enough to find a copy.

† *Lancet*, April 19, 1890.

The members of the Medical Officers of Schools Association, who had excellent opportunities for investigating the subject, came to a diametrically opposite conclusion, and most of them believed that contagion played a great part in the spread of the disease.* In a valuable paper which was published in the xlivth volume of the *Practitioner*,† Dr. Delépine recorded cases which pointed strongly to direct infection as a cause of spread of influenza, and gave a careful analysis of the numbers of the cases which occurred at Broadwood's Piano Manufactory, and the dates on which the patients were seized with the disease.

Several carefully observed cases which point the importance of contagion in the spread of influenza have been recorded by French observers. Dr. Danguy des Déserts‡ reports that an outbreak on *La Bretagne*, a training-ship at Brest, immediately followed the exposure of the crew to contagion from an officer who went on board suffering from the disease. Professor Grasset§ has also shown, on the authority of Dr. Bordone, of Frontignan, that the origin of the outbreak in that town could be traced to infection from a merchant who returned from Paris suffering from the disorder, and who gave a dinner-party to ten people, five of whom were seized with the disease within two days. These were the first sufferers in Frontignan. Dr. Védel, of Lunel, communicated to Professor Grasset‖ an account of the origin and spread of influenza in that place, and Professor Bouchard has put on record the observations of Dr. Tueffart, of Montbéliard. These teach the same lesson. I think, however, that I have said enough to show that the disease is contagious.

With regard to the spread by parcels there is less evidence. Dr. Mease wrote in 1782,¶ "I have no shadow of doubt that the disorder was contagious, and am certain that I myself received the infection from a small trunk of wearing-apparel which came from Dublin where it then raged. I may add that this was the first introduction of it into the town."

Dr. Danguy des Déserts** has recorded a case pointing

* *Brit. Med. Jour.*, May 3, 1890.

† "Is Influenza a Contagious or a Miasmatic Disease?"

‡ *Semaine Médicale*, 1890, p. 5.

§ *Ibid.*, p. 4.

‖ *Leçons sur la Grippe de l'Hiver*, 1889-90, p. 31. Par le Professeur Grasset. Montpelier, 1890.

¶ *Medical Communications*, vol. i, p. 56.

** *Semaine Médicale*, 1890, p. 5.

strongly to the conclusion that infection was conveyed by means of a parcel of goods from Paris to an officer at Brest.

More evidence on this point is much needed, and I should be very grateful for any fresh information on the subject. The matter is of great importance, for if the spread by goods were distinctly proved it might explain some of the outbreaks which have occurred on ships at sea.

To sum up the conclusions at which I have arrived :—

1. There is no doubt that the disease is contagious.
2. Isolated cases precede a general infection of the community.
3. The part played by contagion in the spread of the disease is most important.
4. There is some evidence in favour of spread of influenza by goods.
5. Influenza is not spread in England by a preliminary "contamination of the atmosphere", but the atmosphere is contaminated by individuals who suffer from influenza.